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The Journal of Bone and Joint Surgery

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EDITORIALS AND ANNOTATIONS

SCOLIOSIS

A study of the history of scoliosis is remarkable for the stubbornness with which through the ages this deformity has defied explanation. Since the days of Hippocrates the great students of skeletal deformities have devoted much attention to it, but, as that admirable orthopaedic historian Bick rightly points out, no significant advance on the views of the Hippocratic era was made between Galen in the second century and Glisson in the seventeenth. It was Nicholas Andry who in the eighteenth century expressed the opinion that scoliosis was due to imbalance of the vertebral and paravertebral muscles during the growing years of life. If to-day we add imbalance of the abdominal and limb-root muscles we have said as much as we really know about the pathogenesis of this puzzling deformity. Muscle imbalance is a self-evident cause of scoliosis in anterior poliomyelitis and some other affections of the central nervous system. But in the largest group—idiopathic scoliosis—it has been impossible to establish direct evidence of muscle abnormality, impossible to account for the predominance of the deformity in girls, and impossible to explain why the characteristic patterns of paralytic and idiopathic curves are usually so readily distinguishable. As McFarland suggested recently in opening the discussion on this subject at the Sixth Congress of the International Society of Orthopaedic Surgery and Traumatology at Berne, it may well be that there are different causes for different curves, and several factors in any one case. Somerville seems to have established experimentally that failure of growth of the posterior elements of a segment of the spine is one causal factor. It will be interesting to see if his proposed treatment—partial laminectomy—helps. If it does, it will be almost the first useful preventive measure in the management of the progressive deformities of infancy and childhood which result in such severe and disabling dwarfism. In spite of the beautiful experimental work on the vertebral epiphyses of dogs by Nachlas and Borden, there is little evidence yet that growth on the convex side of the curve can be controlled adequately in the human by any reasonable operative procedure.

It is sad that the cause and prevention of scoliosis so obstinately elude us. By way of compensation it is interesting and fascinating to observe the developments in other aspects of this complex problem. Important information about the behaviour of certain curve patterns has emerged from the reviews of Ponseti and Friedman, of James, and of Stagnara and Queneau. By correlating the site of the primary curve and the age of onset a very fair estimate of the eventual degree of curvature can be made—for example, over 90 per cent of lumbar idiopathic curves developing after the age of ten years do not produce sufficient deformity to justify operation, though fusion for the relief of pain in later life may be necessary; thoracic curves at all ages have a bad prognosis, more than 60 per cent having developed severe deformity at the end of growth; combined thoraco-lumbar curves—the so-called double

primary, four-curve pattern—have an excellent prognosis, seldom producing a cosmetic blemish of significance. If further study confirms that we are able with confidence to visualise the likely end-result, decisions about treatment, and the treatment itself, will be greatly simplified. It may eventually be possible always, as it now is sometimes, to advise correction and spinal fusion at a stage when this is easily done rather than wait until deformity is established and difficult or impossible to correct.

Concerning both conservative and operative treatment ideas have become more clarified. We owe much to the great pioneers of conservative therapy. The list of them is formidable: Hippocrates, Galen, Paul of Aegina, Ambrose Paré, Glisson, and within the memory of some of us, Sayre, Bradford, Lovett, Mackenzie Forbes, Abbott, Haglund, Schanz, Schulteiss, Galeazzi, Schede, Steindler and McCrae Aitken—and this is by no means all. There is unfortunately a tendency nowadays to adopt a defeatist attitude towards conservative treatment, but it still has a place in the properly selected case. In infants corrective beds of the Schede and Denis Browne pattern are useful, combined with baby gymnastics. In older children most of us would agree that asymmetrical exercises and most back supports are valueless. General postural and breathing exercises, periods of rest in recumbency and the maintenance of physical fitness will—to say the least—benefit the patient's health, though admittedly they will not prevent deterioration in a ruthlessly progressive curve. There is increasing evidence that the Milwaukee brace incorporating the age-old principles of distraction and lateral pressure—but continuously and with the patient ambulant—can prevent increase in deformity and can correct established deformity. In the hands of its designers, Blount and Schmidt—two of America's soundest thinkers and workers on scoliosis—this is an admirable brace. In this country difficulties in fitting and in construction have been encountered which it is hoped will eventually be overcome. The conservative measures—distraction plaster-of-paris and Abbott jackets with a vigorous gymnastic routine—used by Stagnara of Lyons are showing promising results. Thus though it may be the case that some curves of good prognosis do well with or without treatment while some of bad prognosis progress in spite of conservative treatment, it seems unwise at this stage in the story of scoliosis to regard conservative treatment as futile. If a combination of exercises, rest and a wearable brace can prevent increase of deformity—and Blount and others have demonstrated that this is possible—until there is no danger of recurrence at the finish of spinal growth, this is surely preferable to a major surgical undertaking in the majority of patients.

Operation is advisable in a small proportion of patients. It is indicated for the rapidly increasing curve, for many severe established deformities, for instability so severe that the patient is unable to sit upright as in some patients with poliomyelitis, and for the relief of pain in the older patients. There have been great advances in operative technique since the early nineteenth century when the first operation for scoliosis was performed by the French surgeon Guérin, who tenotomised the contracted erector spinae on the concave side of the curve. The impetus to new endeavour was undoubtedly given by Hibbs and his co-workers Risser and Ferguson, who between 1914 and 1919 had treated fifty-nine patients with scoliosis by spinal fusion, after correction by traction or by turnbuckle jacket. To Risser must be given the credit for much thoughtful work on scoliosis—work which happily still continues. Indeed it is entirely to our North American colleagues that we owe the gradual development of the surgical treatment of scoliosis—notably to Hibbs, Risser, Ferguson, Mackenzie Forbes, Cobb, Von Lackum, Kleinberg, Blount, Lowman and Mayer. In brief, they have shown that the curved spine can be straightened by various types of jackets, and the correction maintained—or partly maintained—by grafting the primary curve. Sometimes pelvic tilting must be overcome by soft-part operations and the abdominal wall strengthened by fascial grafting. Spinal fusion is more certain if the operation coincides with the end of spinal growth—usually between the ages of fifteen to eighteen years. With modern anaesthesia and blood transfusion shock is no longer a serious problem. Almost any length of the spine

can be grafted at one sitting, especially if bank bone is used; though in this country there are increasing doubts about the reliability of stored homogenous bone, which from time to time seems to absorb, fails to incorporate with the host, or reverts to connective tissue. With a meticulous technique, ensuring that in particular the facet articulations are rawed, pseudarthroses in the grafted area are less likely to occur.

Nonetheless there are legitimate criticisms of this well established procedure: the period of in-patient hospital treatment and of immobilisation is long and costly; it can interfere harmfully with general education; a certain amount of correction is often lost, usually at changes of splinting; correction is sometimes more apparent than real; an appreciable number of pseudarthroses occur; and even successful fusion does not appear to prevent recurrence of deformity under ten years of age. In recent years attempts have been made to obtain correction more quickly and to hold it more securely.

Examples of these efforts are described in this issue of the Journal by Allan and by Roaf—the former using an internal turnbuckle and bone-grafting, and the latter a spinal osteotomy. Neither makes extravagant claims, and the writer of this editorial can pay tribute to the efficacy of both methods performed by their originators on selected patients from his own scoliosis clinic. In the last number of this Journal Professor McFarland, supporting a similar plea from Dr A. R. Shands in his presidential address to the American Orthopaedic Association, issued an editorial warning against experimental surgery which we should all—young and old—do well to heed. The procedures of Allan and Roaf must be regarded as preliminary reports until more time has elapsed and more patients are available for study. But it is proper that preliminary reports of this kind should be recorded in our Journal—our official mouthpiece—so that responsible surgeons throughout the world may be aware of trends and evolutions in the orthopaedic practice of the English-speaking world. Nonetheless it would be singularly unwise for those inexperienced in the operative treatment of scoliosis to embark on these procedures without adequate apprenticeship—and certainly not on lateral spinal osteotomy until they had mastered the steps of the antero-lateral approach to the spine described so clearly by Capener in the Journal of May 1954.

These two operations should be regarded as further steps, promising but also highly specialised steps, in the evolution of the management of the most stubborn deformities. There is not yet sufficient proof that either method is likely to replace completely the more traditional treatment which has proved its worth for so long in the average case of scoliosis requiring operation, and of which a Research Committee of the American Orthopaedic Association reported in 1941: "correction by the turnbuckle jacket and subsequent fusion has yielded better results in this series than have other types of treatment." By and large this is still the case.

H. Osmond-Clarke.

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A.B.C. CLUB OF ORTHOPAEDIC SURGEONS

We do not like the use of capital letters for international or local associations but whether we like it or not the term A.B.C. Club has been adopted to represent the association of American and British members of the English-speaking world of orthopaedic surgeons whose first joint endeavour arose when thirteen young men went from Britain to the United States and Canada in 1948, and when in the next year ten American and five Canadian orthopaedic surgeons came to Britain. This was the beginning of an inspiration based upon the belief that interchange of knowledge between the young surgeons of the various countries of the English-speaking world would advance the cause of surgery, and experience so far has confirmed the soundness of that early view. There is no doubt whatever that the first group of orthopaedic surgeons who went to New York, Philadelphia, Wilmington, Baltimore, Washington, Quebec, Montreal, Toronto, Ann Arbor, Chicago, Detroit and Rochester came back to Great Britain with their minds broadened; and we think that the young American orthopaedic surgeons who travelled throughout England, Scotland and Wales also gained in their outlook. This arrangement of interchange between the continents has now been extended to include the whole of the British Commonwealth as well as the United States of America so that quite recently surgeons of the United States and Canada received a group of Travelling Fellows from England, Scotland, Wales and Australia—and within the next few weeks we in Britain are expecting to receive a similar group of surgeons from the United States and Canada.

These surgeons selected in the United States, Canada, Australia, New Zealand, South Africa and Great Britain do not easily achieve the honour of an award as a Travelling Fellow in Orthopaedic Surgery, but having done so cherish it so much that the A.B.C. Club has been formed which meets once a year. The most recent meeting of the Club was in the Council Room of the Royal College of Surgeons of England in October 1954, a report of which will be found in other pages of the Journal, written by the secretary, Mr F. G. St Clair Strange.

EDITOR.

OSTEOARTHRITIS OF THE HIP*

A Study of the Clinical Pathology

G. C. Lloyd-Roberts, London, England

Lately Nuffield Fellow in Orthopaedic Surgery at the Institute of Orthopaedics, Royal National Orthopaedic Hospital and St George's Hospital

Two aspects of osteoarthritis of the hip will be discussed in this paper. I shall first try to relate the symptoms with the morbid anatomy of the disease and secondly consider some of the factors that predispose towards its development.

Weichselbaum (1872) was the first to recognise that degenerative joint disease could be separated from the other articular disorders. In 1890 Garrod introduced the term osteoarthritis, a synonym which has remained in general use in this country ever since. This final recognition of osteoarthritis as an entity followed earlier accounts of such features as phalangeal nodes (Heberden 1802), eburnation (Bell 1824), " non-suppurative cartilage ulceration " (Adams 1839) and fibrillation (Todd 1843). The observation that the greatest destruction of cartilage and bone occurred in those areas of the articulating surfaces normally in contact (Hein 1858) supported the supposition that the disease was due to excessive wear and tear (Ecker 1843).

Nichols and Richardson (1909) made a full study of the morbid anatomy of osteoarthritis and rheumatoid arthritis and accurately described the characteristic changes in each. Good general accounts of the disease have also been written by Pommer (1913), Allison and Ghormley (1931) and Collins (1949). It is seldom that more than passing reference is made to the changes that occur in the soft tissues within and around the joint. These are usually regarded as " coincidental " (Axhausen 1923, Heine 1926). Some writers have, however, regarded the synovial changes as the primary fault brought about by either infection (Volkmann 1882) or vascular insufficiency (Cecil 1930).

Throughout this period, during which our knowledge of the morbid anatomy of this disease has been advancing, progress has also been made in our understanding of the functional anatomy and physiology of joints. This subject has been recently reviewed by Gardner (1950). It is especially important to emphasise that joint degeneration is a normal part of the process of aging and that this degeneration should not be called osteoarthritis unless it causes symptoms (Heine 1926, Keefer *et al.* 1934, Bennett *et al.* 1942).

THE MORBID ANATOMY OF OSTEOARTHRITIS AND ITS RELATIONSHIP TO THE SYMPTOMATOLOGY OF THE DISEASE

THE SIGNIFICANCE OF SYNOVIAL AND CAPSULAR CHANGES

There are four possible structures from which pain may arise in diseased joints: articular cartilage and underlying bone, capsule and synovial membrane, the surrounding muscles, and (in the hip joint) the periosteum investing the intracapsular bone.

The articular cartilage is known to be insensitive, but some doubt exists about the subchondral bone. The intra-osseous blood vessels are copiously supplied by nerve fibres which are believed to be in part vaso-sensory (Bazett and M'Glone 1928). Under local anaesthesia subchondral bone can be shown to be sensitive but the sensation produced by needle puncture is from scattered points and of a " pressing " or " tapping " nature (Kellgren 1939). Pain may also arise within the muscles controlling the joint, possibly in association with reflex muscle guarding; but the mechanism has yet to be demonstrated.

* An abstract of the Robert Jones Prize Essay 1953.

The articular capsule is, however, known to be sensitive to a variety of stimuli, especially stretching and pulling (Bichat 1799, Gardner 1950, Kellgren and Samuel 1950). There is some doubt about the sensibility of synovial membrane, some holding that it is directly sensitive to painful stimuli (Lennander 1906, Davies 1945), whereas others believe that such pain is usually caused by transmission of the stimulus to the sensitive joint capsule—a mechanism that would be favoured by the subsynovial fibrosis of osteoarthritis. Periosteum, which is a sensitive structure, is found within the hip joint but is seldom involved directly in degenerative disease. It merges, however, with the inner reflected layer of the joint capsule and may be affected if a fibrotic and shortened capsule is subjected to a stretching force.

In the discussion that follows the skeletal abnormalities will be considered in relation to their possible effects upon the joint capsule. While recognising that there may be other sources of pain within and around diseased joints the writer prefers to consider only the joint capsule as a source of pain until the claims of other tissues are more firmly established.

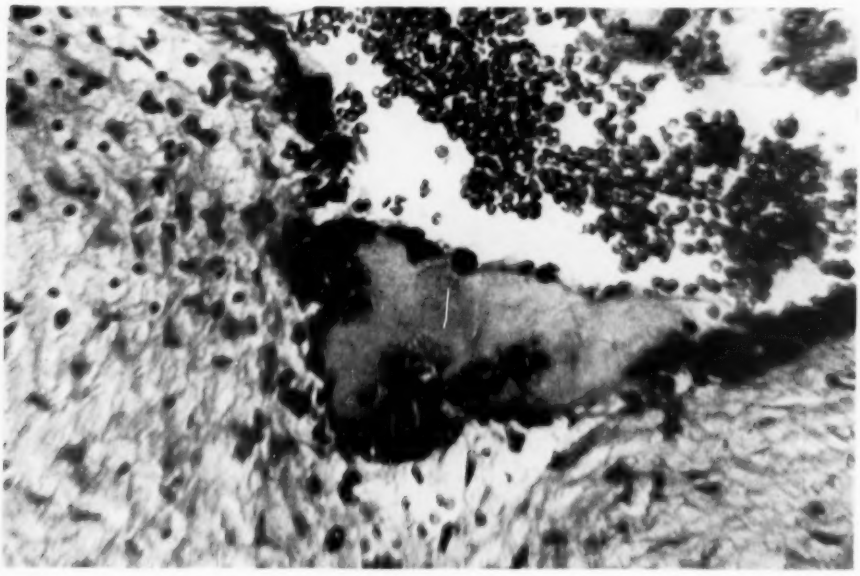

FIG. 1

Section of synovial membrane showing fragment of cartilage which is larger than that usually found. The group of red blood cells indicates relative sizes. (× 350.)

The morbid anatomy and pathogenesis of the synovial and capsular abnormality in osteoarthritis has already been discussed in a previous paper (Lloyd-Roberts 1953) and only a summary will be submitted here. Clinical, operative and histological study of the synovial membrane and capsule in twenty-five patients suffering from osteoarthritis of the hip of sufficient severity to require operation has led to the following conclusions.

1) The synovial membrane is abnormally congested and villous, and the capsule and subsynovial connective tissue are fibrotic.

2) The subsynovial and capsular fibrosis is a response to sustained synovial hyperplasia and congestion (Key 1929, 1932).

3) In osteoarthritis both the synovial hyperplasia and capsular fibrosis are due very largely to the presence of articular debris which can be seen lying beneath the surface layer of the synovial membrane. The debris arises from the degenerate joint surfaces and is removed by the synovial membrane (Hulten and Gellerstedt 1940) (Fig. 1).

4) Gravity appears to direct most of these fragments to the synovial pocket below the neck of the femur so that it is usual to see the greatest amount of synovial congestion in that area. It is probable that capsular fibrosis and shortening begin there, though they later spread to involve the entire capsule of the joint.

5) Many of the symptoms and signs of osteoarthritis of the hip are due to this fibrosis, which interferes with the normal function of the joint capsule. Walmsley (1928) has shown that the whole capsule is tight in the position of extension with some abduction and medial rotation. This is the position of greatest stability on weight bearing. Shortening of any part of the capsule will suppress this position and at the same time expose the capsule to painful stretching produced by the force of body weight. If this shortening occurs inferiorly not only will extension be inhibited but also (for anatomical reasons) medial rotation and abduction. Further shortening will cause deformity towards the position of flexion, adduction and lateral rotation.

6) This deforming property of the capsule is reinforced by reflex guarding in the muscles supplied by the motor branches of sensory nerves to the lower part of the joint cavity—such as the pectineus, quadratus femoris and the adductor group. This dynamic deformity is later followed by a structural deformity from fibrosis at the musculo-tendinous junctions (Steindler 1951).

7) Capsular fibrosis by preventing the hip joint from extending (in which position there is the largest area of femoral articular cartilage in contact with the acetabulum) may tend to speed deterioration in a degenerate joint.

THE MORBID ANATOMY AND CLINICAL SIGNIFICANCE OF SOME OF THE SKELETAL CHANGES IN OSTEOARTHRITIS

Except in detail there is little to add to the many good descriptions of the morbid anatomy of this disease. The observations that follow are based upon the study of thirty-five

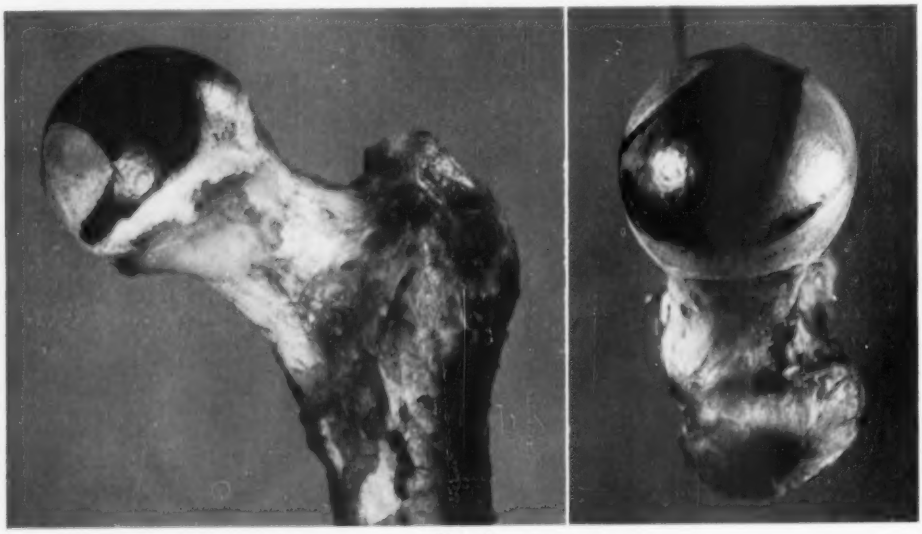

FIG. 2

The upper end of the femur showing in black the area that is normally in contact with the acetabulum during weight bearing.

osteoarthritic femoral heads examined at the time of operation and after excision. The gross specimens were studied in association with pre-operative radiographs and the microscopic preparations with micro-radiographs.

The observations made upon the relationship between individual lesions and symptoms result from a clinical study of 131 osteoarthritic hip joints subjected to operation; this figure includes the thirty-five femoral heads subjected to pathological investigation. The micro-radiograph has proved indispensable in the identification of the precise source of the histological preparations and their orientation with the specimen as a whole. One acetabulum

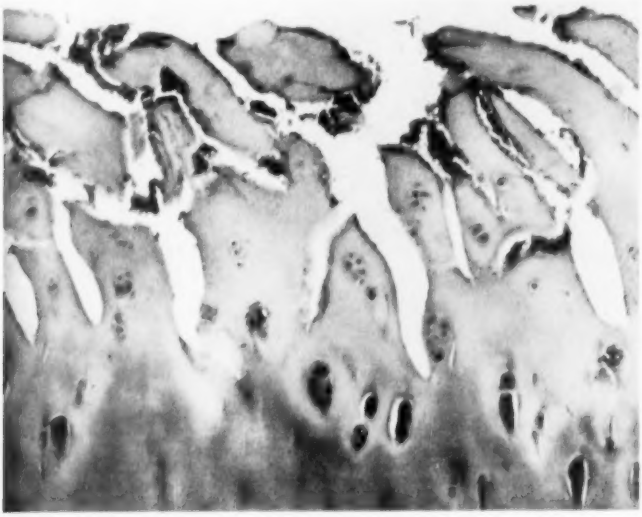

FIG. 3

Articular cartilage in osteoarthritis. Note transverse and horizontal fissuring and tenuous connections of the fragments produced. In superficial area the cells are scanty and the matrix does not take on stain as darkly as the deeper layers. (× 100.)

has also been examined. This was removed at necropsy from a patient who died after an operation during which it is known that the acetabulum was not refashioned. Care was taken in selecting these specimens to exclude cases of osteoarthritis th.' may have arisen from infection, rheumatoid arthritis or avascular necrosis. A few normal hip joints of varying age were also examined so as to familiarise myself with the histological and micro-radiographic appearances in symptomless joints.

Articular cartilage—Most authorities are agreed that the articular cartilage shows the earliest evidence of joint degeneration and that, by the time this has progressed sufficiently to cause symptoms, the greatest loss is found to have occurred in that area of the femoral head on the upper quadrant which is in contact with the acetabulum when the joint is bearing weight or moving within a moderate range (Fig. 2) (Nichols and Richardson 1909, Bennett *et al.* 1942, Collins 1949). This area has been aptly named " the pressure area " by Harrison *et al.* (1953). These workers have also put forward the novel suggestion that the earliest degeneration is to be found in the non-pressure area, and speculate that activity may be necessary for the maintenance of adequate cartilage nutrition—a concept that is supported by animal experiment (Ingelmark and Ekholm 1948). My material does not allow me either to comment on this suggestion or to describe the early changes that occur in degenerating cartilage, such as opacity, blistering and fibrillation. At a later stage, however, the surface of the articular cartilage becomes fragmented, the matrix having first lost its capacity to take up the usual blue tinge with metachromatic stains. Such fragments also show an increased fibrillar pattern and the chondrocytes are lost, scanty or abnormal (Fig. 3).

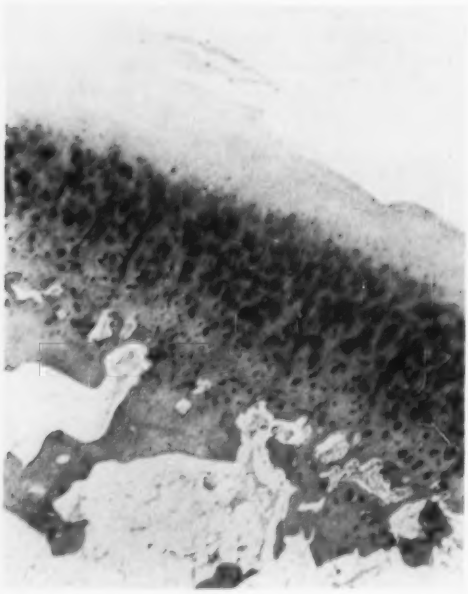

FIG. 4

FIG. 5

Figure 4—Vascular granulation tissue invading subchondral plate. The cartilage is calcified superficial to this. (×22.)
Figure 5—Microradiograph. Note duplication of subchondral line in this area, due to changes shown above.

FIG. 6

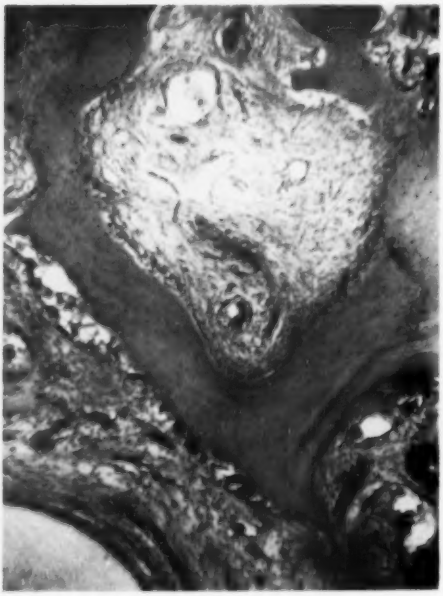

FIG. 7

Figure 6—The remodelling process. Vascular granulation tissue fills the marrow spaces, and bone is being removed by osteoclasts. (×110.) Figure 7—The remodelling process. The marrow spaces are filled with vascular granulation tissue. The trabecula crossing the centre of the field is undergoing bone resorption by osteoclasts below, while osteoblasts are laying down bone above. (×65.)

While these changes are taking place on the surface of the articular cartilage the deeper layers are undergoing modification: cracks extend from the surface as far as the subchondral plate, the cells enlarge, and there is widening of the normal basal layer of calcified cartilage. Pommer (1913) first described the passage of vascular channels through the subchondral plate which is so commonly seen. Some of these channels are filled with granulation tissue, which seems to initiate the formation of new bone superficial to the subchondral plate (Figs. 4 and 5). *Clinical significance*—1) The fragments of cartilage become detached and are subsequently absorbed within the synovial membrane. This causes synovial hyperplasia and later capsular fibrosis. 2) The widening of the calcified zone of the cartilage and the formation of new bone superficial to the subchondral plate account for the "subchondral sclerosis" seen in the clinical radiograph in the early stages of osteoarthritis.

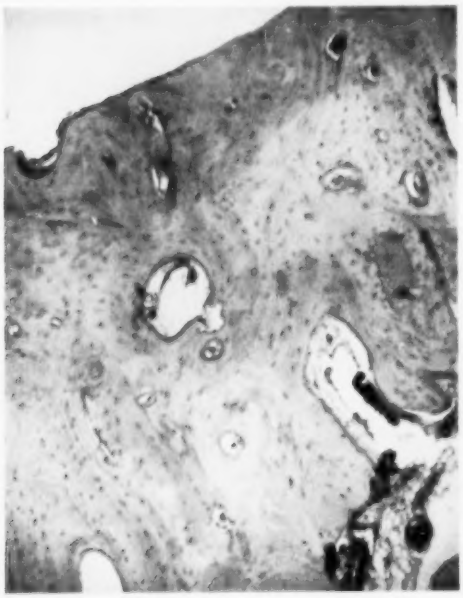

FIG. 8

FIG. 9

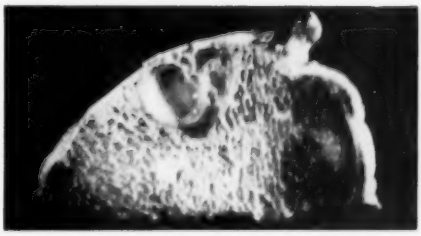

FIG. 10

Figure 8 shows eburnated bone with obliteration of marrow spaces except in a few places. Note that the spaces contain blood vessels. (×65.) Figure 9—Microradiograph. To show area from which section is taken. Note the radiological density of the area. Figure 10—Photograph from same case showing thickening of bone trabeculae extending over a considerable area.

Subchondral bone—Collins (1949), having observed total cartilage loss without subchondral reaction, postulated that this may take an unspecified time to develop. Nevertheless in the cases studied here subchondral changes were well advanced in all cases. Initially the subchondral bone becomes more vascular and granulation tissue fills the spaces between the bone trabeculae in the area below the greatest cartilage damage. Some of this granulation tissue passes through the subchondral plate, but for the most part it seems to be concerned with bone remodelling through the activity of the osteoblasts and osteoclasts which it contains (Figs. 6 and 7) or by metaplasia. Finally the cancellous bone of this area is replaced by dense vascular bone (Figs. 8 to 10).

The cause of these striking changes is obscure, but it seems probable that they represent the reaction of bone, deprived of the protection of articular cartilage, to the stress of weight bearing and activity. A similar reaction may be seen above and below a pseudarthrosis. Bennett *et al.* (1942), however, suggested that the granulation tissue follows fractures of the

subchondral plate, and Luck (1950) supported this view. Although convincing evidence of these fractures was not seen (except when the bone was weakened by cysts) the granulation tissue is like that seen in healing fractures.

This dense (eburnated) bone is formed only when there is severe cartilage damage or complete and localised cartilage loss, and is usually laid down in such a way that the normal spherical surface is not restored because of a loss of bone in the weight-bearing segment. This is probably due to wear and tear at a time when new bone is being laid down and consequently has less resistance to stress. In spite of the apparent toughness of eburnated bone it is evidently less well adapted to its function than articular cartilage, for it can often be observed to continue to wear away if radiographs are repeated.

Clinical significance—1) Radiographic evidence of extension of subchondral sclerosis into the substance of the head below the weight-bearing segment indicates severe cartilage damage. A similar appearance is often seen in the acetabulum. The process does not indicate avascular necrosis. 2) Flattening of the spherical contour of the head and acetabulum in the weight-bearing area indicates that bone debris has entered the joint. 3) There is a predisposition to subluxation, the clinical significance of which is discussed later.

The cysts in osteoarthritis and secondary subluxation—Cysts were found in every one of the femoral heads examined in the area where the cartilage has suffered the most. In consequence they lie within that part of the subchondral bone which is eburnated or in which new bone is being laid down. They frequently communicate with the surface through narrow channels which are visible as pits on the surface of eburnated bone. The channels sometimes contain fibrous tissue continuous with the main cavity of the cyst which spreads to cover the surrounding articular surface and may become modified to resemble fibrocartilage (Figs. 11 to 16). It is tempting to speculate that this represents an attempt at repair. At an earlier stage they contain granulation tissue like that which fills adjacent marrow spaces. The cysts are formed at the expense of the intervening trabeculae. This granulation tissue becomes less vascular and more fibrous until a central area of necrosis surrounded in turn by fibrous tissue and sclerotic bone is formed (Fig. 17). They sometimes contain a jelly-like substance which is not unlike myxomatous tissue. This substance consists of oedematous fibrous tissue. Metaplasia of the fibrous tissue to form fibrocartilage and bone is also seen. Mature cysts sometimes contain necrotic bone fragments which have been detached from the articular surface no longer supported by subchondral bone (Figs. 18 and 19).

Collins (1949) believes that the cysts arise as a result of a failure in the remodelling process, so that osteoclasis outstrips osteoblastic activity in certain areas. The reason for this is obscure, but the necrotic debris that is sometimes seen suggests the possibility of a local vascular fault. Landells (1953), on the other hand, suggested that the cysts are due to the pumping action of synovial fluid which enters the bone through cracks in the subchondral plate, the cracks subsequently becoming clearly defined as the communicating channels. Harrison *et al.* (1953) supported this hypothesis.

My observations favour Collins's view. In support I submit the experimentally produced bone cyst shown in Figure 20. I am indebted to Mr Lipmann Kessel for this illustration, which shows granulation tissue filling a bone cavity that appeared in the femoral head of a guinea pig whose joint had been injected with iodine six weeks previously to destroy the articular cartilage. At this early stage there is no sign of a fluid-filled cavity. The resemblance between this cyst and that illustrated in Figure 11 from a case of osteoarthritis should be noted.

The capacity of cysts to heal spontaneously must be questioned. They may certainly disappear when the surrounding bone collapses, but no convincing evidence of spontaneous healing of a well developed cyst has been seen in radiographs, though this may often seem to happen if succeeding films are of varying penetration.

Clinical significance—Before discussing this subject it is necessary to describe briefly a method of clinical assessment that has been used in eighty-two patients suffering from unilateral

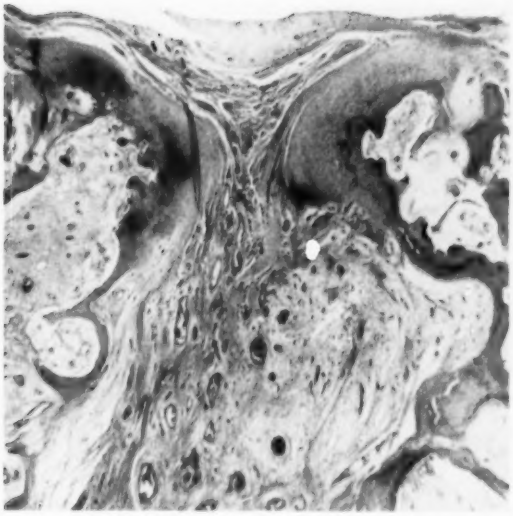

FIG. 11

FIG. 12

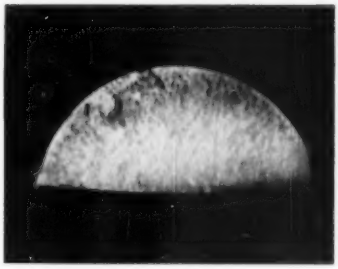

FIG. 13

Figure 11—Section of cyst communicating with surface of femoral head. (×110.) Figure 12—Microradiograph to indicate position of section shown in Figure 11. Figure 13—Photograph showing a femoral head with communicating cyst.

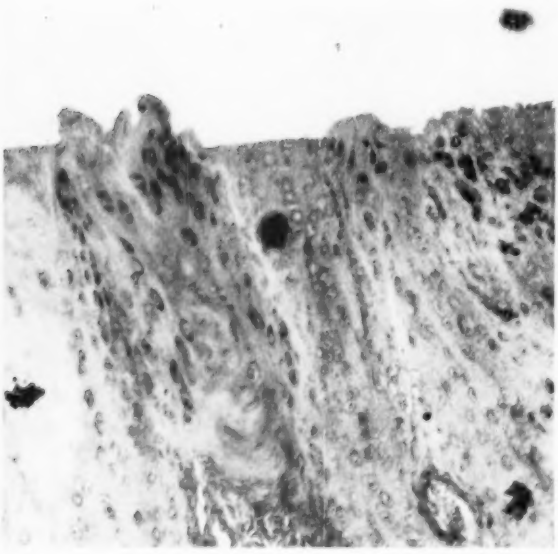

FIG. 14

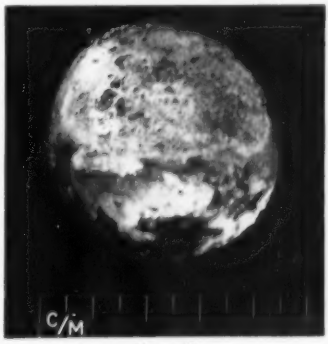

FIG. 15

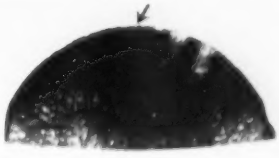

FIG. 16

Figure 14—Section showing the remains of a bone cyst which is still communicating with the surface and is surrounded by dense eburnated bone. (×80.) Figure 15—Photograph of femoral head showing craters on the surface from which articular cartilage has been lost. These craters represent the openings of bone cysts. Figure 16—Microradiograph to show position of section. Two larger cysts communicating with the surface are also visible.

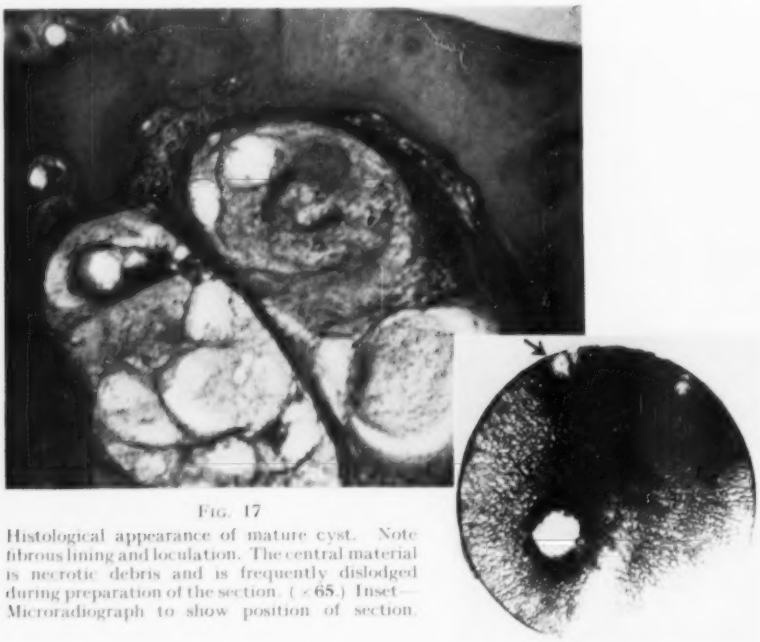

FIG. 17

Histological appearance of mature cyst. Note fibrous lining and loculation. The central material is necrotic debris and is frequently dislodged during preparation of the section. (×65.) Inset—Microradiograph to show position of section.

FIG. 18

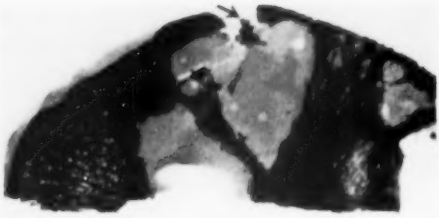

FIG. 19

Figure 18—Necrotic bone in mouth of bone cyst. (×40.) Figure 19—Microradiograph to show site of section.

osteoarthritis requiring operation. Following the recent tendency to grade disability on a system of points (Gade 1947, Muller and Seddon 1953, Merle D'Aubigné and Postel 1954) a method was evolved in which pain and function were considered independently. A normal hip joint would receive sixteen points—eight in respect of freedom from pain and eight for full function. The range lay between nil (one case) and nine points (ten cases), with an average of five and a half points in eighty-two patients whose pre-operative condition had been carefully recorded.

The clinical significance of cysts may be summarised as follows: 1) Cysts visible in the

radiograph usually indicate painful osteoarthritis. This has for long been a clinical impression (Wiles 1949) and receives support from this study. Cysts were seen in the radiographs of seventy-one (88 per cent) in an operative series of eighty-two patients. Plewes (1940) found cysts in only 13 per cent of an unselected group. Table I emphasises this point. 2) Cysts,

FIG. 20

Bone cyst in femoral head of guinea pig after damage to articular cartilage by iodine. Compare this with Figure 11. (×65.)

like eburnation, occur only when degeneration is advanced, which to some extent accounts for their association with painful arthritis. They may also contribute to the amount of joint debris through their surface communications. Their most significant effect, however, is to weaken the articular surfaces so that the hip can subluxate more readily, particularly if the

TABLE I

CLINICAL DISABILITY IN OSTEOARTHRITIS WITH AND WITHOUT CYSTS

Type of case	Number	Average points: pain and function
Osteoarthritis without cysts	11	7·2
Osteoarthritis with cysts	71	5·2
Total	82	5·5

collapse is associated with a previous loss of bone so that the weight-bearing areas of the femoral head and acetabulum are no longer opposing segments of a circle. This has recently been emphasised by Harrison *et al.* (1953). Subluxation is likely to increase the strain upon the already fibrotic capsular ligament, and it is probably for this reason that it is associated with painful arthritis.

Table II compares the degree of disability suffered by patients without subluxation with those in whom subluxation is believed to be primary from dysplasia, and with those in whom it is secondary to the osteoarthritis itself. The striking difference between the primary and

TABLE II

CLINICAL DISABILITY IN OSTEOARTHRITIS WITH AND WITHOUT SUBLUXATION

Type	Number	Average points: pain and function
Idiopathic osteoarthritis with secondary subluxation	23	4·0
Idiopathic osteoarthritis without subluxation	29	5·6
Dysplastic osteoarthritis with primary subluxation	30	5·8
Total	82	5·5

secondary subluxations is accounted for by the advanced stage that the disease must have reached before secondary subluxation is possible in a hip joint with an originally normal acetabulum.

The bony ridge on the femoral neck—Many cases of advanced osteoarthritis show radiographic evidence of new bone formation on the under surface of the femoral neck (Fig. 21). This bony ridge was described by Wiberg (1939) as a buttress formed in response

FIG. 21

FIG. 22

Figure 21—Radiograph showing new bone below neck of femur. Note the laminated appearance and the well formed acetabulum. Figure 22—Section taken from bony ridge running below neck of femur. Note the remodelling that is occurring in the subperiosteal zone. Osteoclasis and osteogenesis in progress. (× 65.)

to altered lines of force in congenital subluxation. The ridge was found by Wiberg to lie behind the coronal plane, an observation with which I agree.

In a previous communication (Lloyd-Roberts 1953) I suggested that this ridge is

due to the deposition of new bone within a subperiosteal space produced by traction transmitted through the fibrotic capsule to the periosteum investing the neck. This traction force will, of course, be greater when subluxation is present and consequently the ridge will more often be found in such cases, but it also occurs when there is no subluxation (Fig. 21). Furthermore it does not appear in congenital subluxation until osteoarthritis supervenes although the abnormal forces have been present since childhood. Figure 22 illustrates the microscopic appearances of such a case at sixty. Active remodelling is still in process although the subluxation has probably been present since birth.

TABLE III

CLINICAL DISABILITY IN OSTEOARTHRITIS WITH AND WITHOUT INFERIOR RIDGE OF NEW BONE

Type	Number	Average points: pain and function
New bone present	46	5·3
New bone absent	36	5·8
Total	82	5·5

Clinical significance—1) The bony ridge occurs in advanced disease and indicates that capsular fibrosis and adhesions are present. 2) Considering again the group of eighty-two hip joints requiring operation we find that new bone is present in more than half the cases and is associated with a slight increase in disability (Table III). 3) Capsular shortening in the inferior part of the joint sufficient to produce this change might be expected to declare itself by a limitation of abduction or an adduction deformity. Table IV shows an analysis of the incidence of fixed adduction deformity in seventy-three patients whose pre-operative examination was sufficiently detailed to warrant their inclusion. There seems to be a significant relationship between the bony ridge and adduction deformity.

TABLE IV

RELATIONSHIP BETWEEN BONY RIDGE AND FIXED ADDUCTION DEFORMITY

Type	Number	Fixed adduction	Per cent
New bone present	32	24	75
New bone absent	41	10	24
Total	73	34	46

Osteophytes in osteoarthritis—Osteophytes are a conspicuous feature of osteoarthritis, and their origins and significance have prompted considerable speculation. They are found either upon that part of the femoral head or acetabulum which is neither concerned with weight bearing nor movement within the habitual range, or as a ring around the junction of the articular cartilage of the head with the neck of the femur. In short, they occur anywhere except upon the upper quadrant of the head and its opposed acetabulum. Harrison *et al.* (1953) have emphasised this and demonstrated the vascular anatomy of the osteophyte, which consists of a central core of cancellous bone continuous with the parent bone and covered on its surface by fibrous tissue or fibrocartilage.

The osteophytes appear to arise either by the extension of the bone into the degenerate articular cartilage where this is not prevented by friction or weight bearing (Pommer 1913)

(Fig. 23) or by metaplasia within the marginal zone where synovium, capsule, bone and articular cartilage all meet (Weichselbaum 1872) (Fig. 24).

Clinical significance—The significance of osteophytes is far less than their conspicuous nature would suggest. There is no evidence that they are concerned in the pain of osteoarthritis. Although often present when movement is severely limited, they are equally often absent. They do not develop in areas of the joint where they would be subjected to weight bearing or frictional stress; so it is unlikely that once developed they will acquire the ability to prevent movement by interlocking growth. They may often appear to do so when seen at operation, but I believe that they are more likely to have developed in this way because movement has been restricted for some other reason such as capsular fibrosis. Fibro-cartilaginous loose bodies

FIG. 23

Section showing subchondral plate and deep cartilage layers left behind by advancing osteophyte. (×32.) Inset—Microradiograph illustrating position of section.

are uncommon. Only two hip joints among 131 showed such bodies in the pre-operative radiograph, and these may well have developed in the synovial membrane by metaplasia rather than by fracture of osteophytes—an event that might be expected to occur more frequently if the osteophytes caused limitation of movement by oppositional growth.

It is possible that osteophytes encourage synovial hyperplasia by abrasion and so contribute to the capsular fibrosis. It is also possible that they indicate the duration rather than any other features of joint degeneration. This latter speculation is supported by their slow development during the progressive joint degeneration that occurs with age (Bennett *et al.* 1942) and their frequent absence when disabling osteoarthritis occurs in association with an obvious abnormality in relatively young people.

The osteophytes that may develop in the floor of the acetabulum when subluxation occurs are (in association with other features) of some diagnostic significance when osteoarthritis complicates congenital subluxation (Wiberg 1939). In reconstructive operations they must be

removed if the refashioned or substituted femoral head is to be restored to its normal position within the acetabulum (Figs. 25 to 27).

DISCUSSION

I have attempted to relate some of the bony and cartilaginous changes that accompany osteoarthritis of the hip with the symptoms of the disease in the light of the concept that capsular changes are secondary to the former and largely responsible for the latter. I shall now try to extend this hypothesis to cover other aspects of osteoarthritis.

It is usually thought that the radiographic and clinical features are poorly related. But if the changes visible in the radiograph are considered in terms of the invisible capsule some of these difficulties are lessened. If osteophytes are ignored, if cartilage and bone debris are estimated from diminution of joint space, and if eburnation and cystic change and the presence of subluxation are noted, some estimate of the probable degree of capsular change—and so the clinical severity of the condition—becomes possible. If in addition new bone is present below the femoral neck and calcification is visible in the capsule above the neck, severe limitation of movement or fixed deformity is likely to be present. By this means some connection will be found to exist between radiographic and clinical features, but in many the symptoms are less severe than the radiograph would suggest. This dissociation is explained in some cases by the advanced age of the patient or some other factor reducing activity to a degree that allows the degenerate hip joint to fulfil its functions. This has been mentioned in a previous paper in a discussion of the relationship between the physiological aging process in joints (Bennett *et al.* 1942) and degenerative joint disease (Lloyd-Roberts 1953) (Figs. 28 and 29).

FIG. 24

To show marginal zone in which the capsule (seen at top of section) joins the edge of articular cartilage seen on right. On the left there is a mass of irregular fibrocartilage which in places is becoming calcified. (×30.)

Patients in whom fixed deformity is severe and the range of movement markedly restricted frequently suffer remarkably little pain in the hip. In these patients the capsule has shortened to a considerable extent and this, together with muscle shortening and bony adaptation, causes deformity and limitation of those movements that are painful. A similar mechanism may well explain the success of McMurray's intertrochanteric osteotomy, in which the proximal fragment takes up a position of adduction and lateral rotation together with some flexion. This is a position of capsular relaxation in which there is less likelihood that capsular strain will develop, provided the distal fragment is in a good functional position (Fig. 30). Such strain will recur and pain may follow if subluxation of the hip follows the osteotomy.

An osteoarthritic joint sometimes becomes irritable and is then often referred to as "hot" or "acute." Various possibilities may account for this. The nipping of synovial fringes is one, but in this series there has been no apparent tendency for those cases with large synovial polyps to be thus affected. The fracture of osteophytes is an unlikely cause

because loose bodies are rare. Synovitis, from a sudden shower of joint debris, can be no more than a speculation but synovitis due to intra-articular haemorrhage has some support

FIG. 25

Osteoarthritis with subluxation of hip. Note osteophyte in acetabular floor. Marked sclerosis of femoral head. New bone formation below neck of femur.

FIG. 26

FIG. 27

Figure 26—Microradiograph of coronal section of acetabulum seen in Figure 25. An osteophyte can be seen arising from the floor of the acetabulum. Figure 27—Coronal section of the same acetabulum. The junction between the osteophyte and the original floor of the acetabulum can be seen.

from observed facts. The knee joint in such a case can be felt to be warm and haemarthrosis or infection may be suspected. Aspiration has yielded blood. Wheeldon (1953) has found blood in similar cases involving the ankle and shoulder. The depth of the hip joint obscures

FIG. 28

FIG. 29

Patient aged sixty-four with bilateral osteoarthritis of hip joints. Figure 28—This joint was never painful. Loss of medial rotation and extension only. Note one cyst only in hip of acetabulum. Sclerosis is subchondral only. Joint space reduced over small area. No subluxation. No new bone below neck of femur. Neutral position. Note osteophytes on head. Figure 29—Painful hip. Arthroplasty performed. Note numerous cysts and sclerotic bone on femoral and acetabular side of joint. Joint space reduced over a wide area. Slight subluxation indicated by presence of new bone in floor of acetabulum. Some new bone below neck of femur. Lateral rotation deformity.

FIG. 30

Radiograph after McMurray's intertrochanteric osteotomy. Note adducted position of upper fragment.

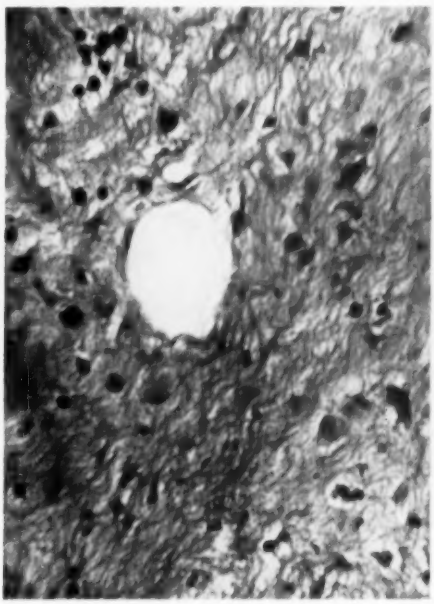

FIG. 31

Haemosiderin granules found within deeper layers of synovial membrane in osteoarthritis of the hip. (×245.)

these signs, but fluid of a chocolate colour is sometimes found at operation and it is not uncommon to find haemosiderin within the synovial membrane in osteoarthritis (Fig. 31).

Lastly, brief reference should be made to osteoarthritis in some other joints. Figure 32 illustrates joint debris within the synovial cavity beneath the phalanx from a case of hallux rigidus. Capsular fibrosis here would limit dorsiflexion provided the toe did not subluxate

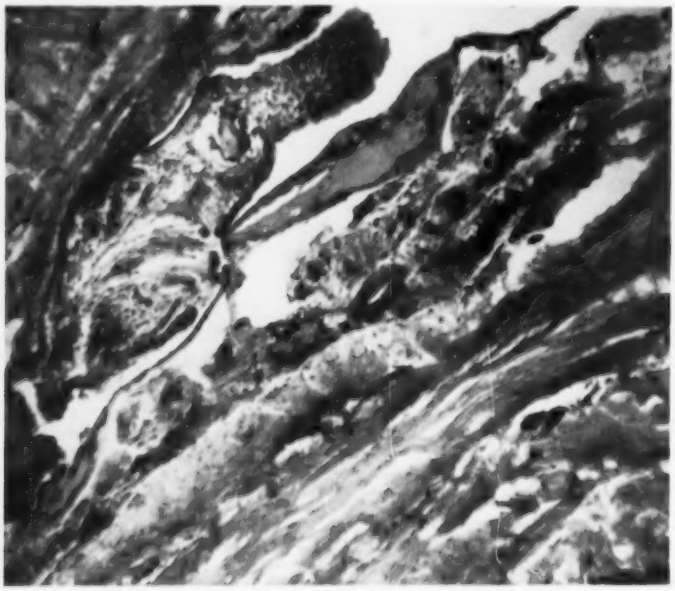

FIG. 32

Hallux rigidus. Synovial membrane and capsule removed from the area of reflection below the proximal end of the phalanx. Note large fragment of cartilaginous debris lying between the two synovial layers, and small fragment at point of reflection at left. (×85.)

into valgus. Osteoarthritis of the shoulder is rare, but when it occurs abduction and lateral rotation are the movements most impaired. Ellis (1952) performed arthrograms on cases of periarthritis and found that the lowest part of the joint cavity was obliterated by adhesions in some cases. Among Ellis's series were some joints in which osteoarthritis was also present, and these too showed similar appearances in the arthrogram, changes that would on anatomical grounds interfere mainly with abduction and lateral rotation. Macnab (1952), investigating the results of operation for recurrent dislocation of the patella, found that operations in which the patella was not excised might stop further dislocation but were less successful in preventing generalised osteoarthritis of the knee. The reason why localised patello-femoral arthritis should spread to involve the tibio-femoral joint is not clear unless it is explained by the presence of joint debris in the main joint cavity, which, arising from the patella, causes synovial changes and later capsular fibrosis. Joint debris has been seen at a distance from the patella in two such cases. In the knee the greatest stability is in extension when the capsular ligaments are tight. Capsular shortening would result in a loss of full extension, with the danger of cartilage damage due to abnormal weight bearing and instability, further capsular change and lastly flexion deformity accentuated by the downward thrust of body weight.

THE ETIOLOGY OF OSTEOARTHRITIS OF THE HIP

MATERIAL

The observations made in the rest of this paper are based upon a study of the cause of osteoarthritis in 131 hips requiring operation. This is a consecutive series from which seven must be excluded because of insufficient pre-operative information. Seven patients had both hips operated upon; so the final analysis is of 124 hips in 117 patients.

The cases were unselected other than by the severity of their symptoms and the exclusion of those with evidence of post-traumatic ischaemia occurring in adult life. The first stipulation excludes those mild cases in which the normal aging process may complicate the clinical and radiological findings, and the second excludes those in which the necrotic changes in the subchondral bone deprive the articular cartilage of nutrients (Ekholm 1951) and mechanical support so that degeneration of articular cartilage is no longer the primary abnormality.

The cases under review were placed in etiological categories, as shown in Table V.

TABLE V

ETIOLOGICAL FACTORS IN 124 CASES OF OSTEOARTHRITIS OF THE HIP

Unknown causes (idiopathic)	73
Dysplasia (primary subluxation)	26
Adolescent coxa vara	8
Perthes' disease	3
Infective (non-rheumatoid)	3
Rheumatoid arthritis	2
Protrusio acetabuli	2
Traumatic dislocation	2
Trochanteric fracture	1
Acromegaly	1
Paget's disease (coxa vara)	1
Caisson disease	1
Syphilis (mild Charcot joint)	1
Total	124
Cases of known etiology	51 (41·2 per cent)
Cases of unknown etiology	73 (58·8 per cent)

Four aspects of the etiology and pathogenesis of the disease will be discussed; first, joint degeneration following some known disease, deformity or injury that has caused incongruity between the joint surfaces; secondly osteoarthritis secondary to congenital dysplasia; thirdly the role of insidious ischaemia; and lastly "idiopathic" osteoarthritis.

INCONGRUITY OF JOINT SURFACES

Incongruity between the articular surfaces of the femoral head and the acetabulum is a recognised cause of osteoarthritis. In this series there were fifteen (12·1 per cent) osteoarthritic hips attributable to this—eight of adolescent coxa vara with uncorrected deformity, three of

Perthes' disease with imperfect reconstruction of the femoral head, two of protrusio acetabuli, and one each of coxa vara complicating Paget's disease and intertrochanteric fracture.

When the articular surface is irregular, as in healed Perthes' disease, the onset of osteoarthritis is readily explained by the analogy of the wearing of a badly fitting moving joint in a machine, and further damage by the " grinding mill " effect of the particles worn away. This, however, is as far as this mechanical analogy should be allowed to extend in describing the development of osteoarthritis of the hip joint, which is so often erroneously regarded as a simple ball-and-socket joint. If the analogy were sound it would be difficult to understand why osteoarthritis should ever follow uncorrected adolescent coxa vara, in which the capital epiphysis remains viable and the articular cartilage undamaged. Such a ball-and-socket joint would compensate for the altered direction of the shaft of the femur by an alteration in its axis of movement, with no increased liability to degeneration. In coxa vara there is little change in the relationship between the femoral head and the acetabulum (slipped epiphysis is a misleading term) but only between the femoral neck and the head (Figs. 33 and 34).

FIG. 33

FIG. 34

Adolescent coxa vara. Figure 33—Before correction. Figure 34—After correction by manipulation. Note that the position of the head in the acetabulum has not changed appreciably.

Nevertheless osteoarthritis does develop as a sequel to coxa vara in spite of apparent viability of the femoral head and its overlying articular cartilage, making discussion of some of the possible causes of cartilage degeneration and osteoarthritis necessary. To do this I shall first describe briefly the mechanism of weight bearing through the hip joint.

The load carried through the extended hip joint when standing on one leg is approximately two and a half times the total body weight. This represents 385 pounds in the case of a man weighing eleven stones (154 pounds) which is a measure of the mechanical disadvantage of this joint (Inman 1947). This ratio will be greatly increased by simple vertical movements such as stepping from the pavement to the road. Steindler (1951) described a " propelling phase " in walking in which the extended hip both carries the body weight and transmits the force necessary for forward movement, a combination which must still further increase the load. This extended position was described by Walmsley (1928) as that in which the hip is most stable, the opposing articular surfaces most close fitting and in contact over a larger area than otherwise, and in which all parts of the capsule are tight. The increasing capsular tension, especially anteriorly, appears to steer the bones into this position which, once adopted, depends in part for its stability upon intact and powerful anterior capsular ligaments. This

position of extension is associated with slight abduction and medial rotation, which not only tilts the opposite side of the pelvis upwards and forwards, so facilitating the next forward step, but also places the femur in a favourable position for locking of the knee by lateral rotation of the tibia. Dissection of a fresh cadaveric joint will demonstrate the stability of this position and the capsular tension associated with it (Figs. 35 and 36). The area of femoral articular cartilage in contact with the acetabulum may also be demonstrated by injection of melted paraffin wax into the extended joint, and is designated "the pressure area" by Harrison *et al.* (1953). As the hip is moved from this position the contact area becomes smaller and the capsule less tense. In the living subject lying prone, stability in extension is demonstrated by a loss of rotary or sideways movement.

Small though the pressure area is, it is the largest available for transmission of these great forces through the hip joint. Its ability to withstand this load may be explained by recent investigations into the structure of articular cartilage in weight-bearing areas of the knee. Matthews (1953) found the ratio of chondroitin sulphate to collagen to be increased here compared with non-weight-bearing areas, and observed that it is reduced when cartilage degenerates. Hirsch (1944) stressed the importance of chondroitin sulphate in maintaining the elasticity of articular cartilage, and Benninghoff (1925) noted that its loss renders articular cartilage more liable to injury and described, in addition, a modification of the fibrillar arches between different areas of articular cartilage. These features suggest that the pressure area may consist of cartilage adapted to withstand exceptional stress.

Lastly there is the lubricating property of synovial fluid, which functions only when the joint is moving and therefore ceases to act when the joint is stationary in the position of stability (MacConaill 1950). Fluid lubrication cannot operate between perfectly congruous joint surfaces. It may, however, assist movement and spreading of the load before this position is reached.

Returning now to the problem of adolescent coxa vara and the normal femoral head lying within the normal acetabulum, it would seem that there is nothing to prevent the head and acetabulum establishing contact in the position of stability. This position of the joint will, however, cause the femur to lie in a position of relative adduction and lateral rotation to a degree which will depend upon the amount of displacement in the neck. If the deformity is excessive walking will be difficult and so the joint will be held in abduction and medial rotation to restore the vertical alignment of the femur. This, in turn, will mean that extension is not possible. Flexion will occur and the stable position will not be used in weight bearing. This results in a loss not only of stability but also of the support of a tight capsule. In addition the area of acetabulo-femoral contact will be both reduced in size and possibly less well adapted in structure.

Although adolescent coxa vara has been specially considered it is probable that a similar mechanism influences the evolution of osteoarthritis in other conditions. Congenital subluxation often leads to osteoarthritis, but the articular cartilage is normal. Here the instability between the skeletal elements may neutralise this mechanism, and anteversion of the neck may cause it to be suppressed if this results in excessive medial rotation of the leg. In Perthes' disease the position may be unattainable because of disproportion between femoral head and acetabulum, and thus an increased strain is placed on an already irregular articular surface. Similarly in "idiopathic" osteoarthritis capsular fibrosis, once established as a result of cartilage wear, may by contracture prevent free extension and so render the already degenerate cartilage more vulnerable to attrition.

It is suggested that the mechanism described accounts for the development of osteoarthritis in many cases where we are accustomed to speak of the "altered mechanics of weight bearing"—an explanation which is both vague and at best only superficially satisfying. To invoke the analogy of a simple and congruous ball-and-socket joint and apply it to the complex functional anatomy of the hip is positively misleading, for it erroneously

FIG. 35

Normal hip joint in extension viewed from below. Note that the capsule is tight.

FIG. 36

Normal hip joint in 10 degrees of flexion. Note that the capsule has now loosened.

presupposes a state which, if it existed, would often prevent degenerative joint changes. It is possible that in this group a means of prevention offers itself through precise correction of deformity in young patients when this can be achieved without prejudice to the viability of the femoral head.

DYSPLASIA OF THE HIP JOINT

Dysplasia is used here to mean a developmental abnormality of the hip which may involve the acetabulum, head of the femur or its neck so as to represent in minor degree the anatomical changes found in congenital dislocation of the hip. The group includes those abnormalities which fall between the stage of " pre-luxatio " (Putti 1933), in which all the features of congenital dislocation are present but the hip has not yet dislocated, to minor degrees of shallow acetabulum, coxa valga, anteversion of the femoral neck and deformity of

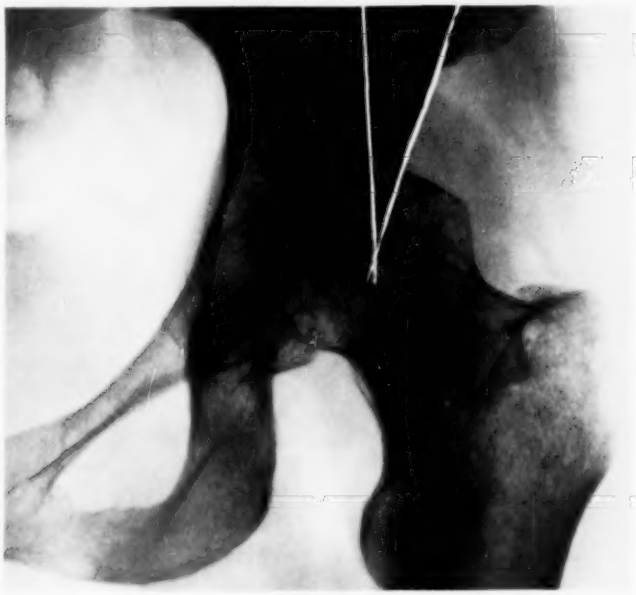

FIG. 37

Early osteoarthritis of the hip probably due to dysplasia. The " CE " angle is outlined and measures 20 degrees. For comparison with Figure 42.

the femoral head. This condition is described as a primary subluxation when the femoral head has moved upwards but still retains its contact with the acetabulum.

Congenital dysplasia is important not only as a cause of osteoarthritis but also because its early recognition presents an opportunity for us to prevent the disease from developing.

In this review of 124 hip joints, osteoarthritis was thought to be due to dysplasia in twenty-six cases (20·9 per cent). The average age at onset of symptoms in twenty-three cases was 46·6 years and the average duration of symptoms before operation was 7·9 years. There were sixteen unilateral and ten bilateral cases and the incidence in females was twice that in the males.

These figures agree fairly closely with those of Wiberg (1939), who found an incidence of 25·7 per cent among unselected out-patients. The average at onset of symptoms was 33·5 years for females compared with 54 years for males. Karlén (1944), however, proposed that 31 per cent of his cases arose upon this basis; Putti claimed approximately 40 per cent and Gade (1947) 47·9 per cent, a figure more than double that estimated in the present series. The conflict between Gade's figure and my own is all the more remarkable because we are concerned with a like number of joints (124 compared with 123) all requiring operation. Gade included thirteen cases following evident ischaemic change which accentuates the contrast.

This difference is probably due to the difficulty of establishing criteria for this diagnosis in advanced cases of osteoarthritis. Wiberg (1939) emphasised this and described features that distinguish this type of osteoarthritis based on a study of eighteen patients in whom

FIG. 38

FIG. 39

To illustrate subluxation occurring in an osteoarthritic hip of unknown etiology over a five-year period. Figure 38—Initial radiograph. Figure 39—Five years later. Note the osteophytes in the floor of the acetabulum.

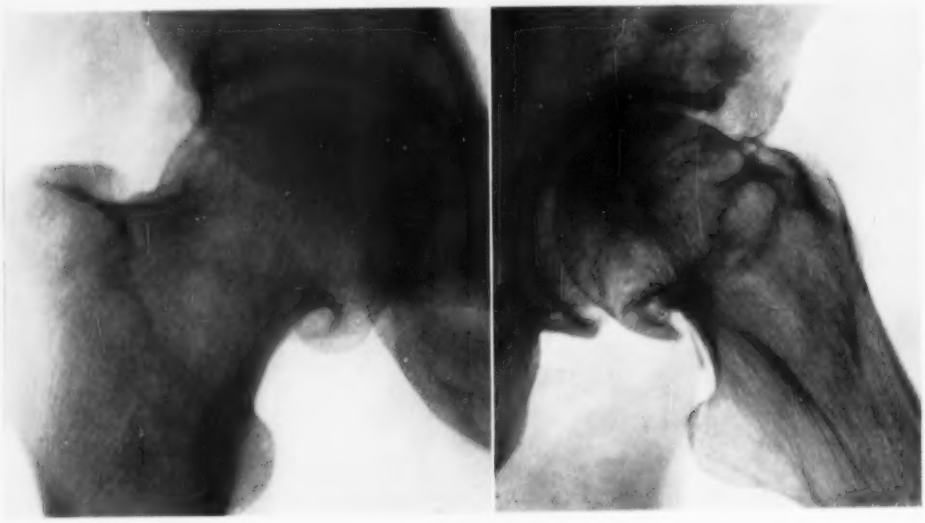

FIG. 40

To show " capital drop " without subluxation of the hip.

he had radiological evidence of mal-development of the acetabular roof before the development of the disease. He measured the angle that a line drawn from the outer and upper edge of the acetabulum to the centre of the femoral head makes with a vertical line drawn at right

angles to the horizontal between the centres of the femoral heads (Fig. 37). He called this angle the " CE " angle which normally measures at least 25 degrees. If it is less than 20 degrees the acetabulum is abnormally shallow and if between 20 degrees and 25 degrees the acetabulum is doubtfully normal. He described three radiological signs that occurred when arthritis followed: First, sclerosis of the acetabular roof, secondly an osteophyte on the lower segment of the head—the " capital drop "—and lastly bone formation in the acetabulum—the " double acetabulum." He also described the bone ridge on the lower border of the neck of the femur, which he suggested developed because of abnormal weight bearing.

All these features may be seen in cases of osteoarthritis secondary to subluxation. Unfortunately, however, they may all be seen in cases of osteoarthritis in which there is no suggestion of an underlying primary subluxation. Acetabular sclerosis is an early sign and merges into the subchondral sclerosis of osteoarthritis of whatever etiology. Acetabular new

FIG. 41

FIG. 42

Figure 41—To show severe osteoarthritis developing on basis of subluxation at age of forty-one. Note butter-shaped head of femur and new bone below neck of femur. The femoral neck is valgoid and there is a large osteophyte in the floor of the acetabulum. Figure 42—Severe osteoarthritis of the hip with subluxation. This hip joint is the same as that illustrated in Figure 37 after an interval of four years.

bone also occurs in association with secondary subluxation (Figs. 38 and 39). The capital drop may be seen in the absence of subluxation (Fig. 40). Bone formation at the lower border of the femoral neck may occur in the absence of significant subluxation (Fig. 21).

There is little difficulty in attributing cases of osteoarthritis to earlier subluxation when the mal-development is severe. Not only is the displacement obvious and Wiberg's sign present, but also the neck is often markedly valgus (or inverted) and the head buffer-shaped (Fig. 41). Minor degrees of dysplasia may be obscured when osteoarthritis develops, but more commonly a secondary subluxation associated with acetabular new bone mimics a primary malformation (Figs. 38 and 39). The capital drop, however, very rarely appears in these cases and is therefore of value as a distinguishing feature; unhappily it is not always present in association with dysplastic subluxation (seventeen out of twenty-six in this series).

There is, for example, no certain method of determining the cause of osteoarthritis with

marked subluxation in the case illustrated in Figures 37 and 42 even though a radiograph taken four years before is available and suggests the possibility of a minor dysplasia (CE angle 22 degrees) without subluxation. The etiology in this case remains uncertain on the radiological evidence but is likely to be acetabular dysplasia. The early onset of symptoms, which began at the age of forty-three, and the absence of involvement of other joints does, however, provide additional support for a dysplastic etiology in this and many other such cases.

The effect of fixed deformity upon the radiological features is also important (Figs. 43 and 44). These radiographs show the appearance of a normal hip joint in neutral position and in flexion, adduction and lateral rotation (the position of deformity in osteoarthritis). There is an illusion of coxa valga and a femoral head poorly supported by the acetabulum.

In my assessment of these 124 osteoarthritic hips I have constantly borne these difficulties in mind, and I am conscious of having adopted a very critical attitude in accepting cases of established osteoarthritis as having a basis in a congenitally dysplastic hip. It is, I think, probable that some of the seventy-three hips (58·8 per cent of the whole group) in

FIG. 43

FIG. 44

Figure 43—Radiograph of a normal hip joint in neutral position. Figure 44—Radiograph of a normal hip joint in flexion, adduction and lateral rotation.

which osteoarthritis has been described as "idiopathic" have occurred as a result of this malformation, though it was not recognisable with certainty when the disease was fully developed. I believe that my figure of 20·9 per cent represents, therefore, a low estimate and that the true incidence may lie nearer to 25 per cent—the figure quoted by Wiberg, whose investigations had given him a clear appreciation of the evolution of osteoarthritis in dysplastic hip joints. Furthermore Wiberg's cases were in out-patients and probably included early lesions; thus the diagnosis could be made confidently in contrast to the advanced cases reviewed here.

Although a shallow acetabulum is probably the most important element, it is possible that associated malformations contribute to the development of osteoarthritis. A buffer-shaped head and a true coxa valga are frequently seen, especially in those cases with unquestionable subluxation (Fig. 41). La Scalen (1924) investigated these features but did not observe them before the third or fourth year of life and so assumed that they were secondary to a shallow acetabulum. Delayed development of the capital epiphysis (Putti 1929)

would make it difficult to assess the shape of the femoral head at an earlier date, but her view regarding the secondary nature of coxa valga is supported by Muller (1927). Kindemann (1933) on the other hand regarded this as a primary deformity in dysplasia and due to the same defective anlage.

Hass (1951) reported the anatomical changes that he found within the hip joints of an infant with markedly shallow acetabula who died shortly after birth. He observed that the cartilaginous structure of the hip socket " corresponded in shape and depth to the normal measurements for the newborn " and concluded that " we have to deal principally with a hypoplasia of the osseous nuclei." In contrast to the acetabulum, the femoral head was smaller than usual and the femoral neck was valgoid. It appears then that both the head and the neck of the femur can be deformed at birth and take part in the primary abnormality, their deformity later becoming more conspicuous as development proceeds. Hass also mentioned that anteversion of the femoral neck was increased in his case from the normal 20 degrees to 30 degrees and later (speaking of the anatomy of fully developed subluxation) he remarked that anteversion is a very common associated feature. This observation is supported by the numerous cases in which anteversion is found when prosthetic replacement operations are performed in osteoarthritis (Merle D'Aubigné 1954).

The radiographs shown in Figures 45 to 47 support the view that coxa valga can exist and persist independently in the presence of a normal acetabulum and be uninfluenced by treatment in an abduction splint.

The balance of evidence, therefore, suggests that deformity of the head, coxa valga, and anteversion are each part of the dysplastic malformation. It is uncertain in what proportion they (as opposed to a shallow acetabulum) contribute towards th development of osteoarthritis. They may, however, affect the normal weight-bearing mechanism of the hip joint in a manner already described in the preceding section.

The condition of the " healthy " side in unilateral congenital dislocation of the hip has been studied by Severin (1941) and by Muller and Seddon (1953). Such " healthy " hips, as these authors point out, are frequently dysplastic and offer an opportunity for observing the development of osteoarthritis. The incidence of dysplasia on the " healthy " side, as found by Severin, was fifty-seven hips out of 190 infant patients. Of these, fifteen showed normal acetabula when examined five to twenty-seven years later and a further ten became almost

TABLE VI

STATE OF "HEALTHY" HIP IN 147 PATIENTS WITH UNILATERAL CONGENITAL DISLOCATION

Adult acetabulum	Number of cases	Original radiographs		Adult radiographs	
		Normal or doubtful	Abnormal	Normal or doubtful	Abnormal
CE less than 20 degrees	17	1	4	0	5
CE 20–25 degrees	25	4	2	6	0
CE more than 25 degrees	105	12	6	18	0
Totals	147	17	12	24	5

normal. In other words thirty-two hips out of the original 190 on later examination proved to be definitely abnormal. This finding resembles that of Muller and Seddon, who found twenty-eight dysplastic hips in a similar series of 172 " healthy " hips followed into adult life.

I have had an opportunity (which I gratefully acknowledge) to examine the records and radiographs of Muller and Seddon's cases which were made at the time of follow-up examination. Among these 172 patients who were re-examined in later life by Muller and Seddon

FIG. 45

FIG. 46

FIG. 47

Coxa valga without dislocation. Figure 45—At age of nine months. Left hip dislocated. Right acetabulum normal. Figure 46—Eighteen months later, after treatment in double abduction hip spicas for that period. Note marked valgus of neck in both hips. Figure 47—At age fifteen. The valgus deformity of the femoral neck has persisted and resembles that on the dislocated side (not shown).

and who, at the time of re-examination, had reached the age of sixteen years, seventeen had definitely abnormal acetabula (CE angle less than 20 degrees), twenty-five were doubtful (CE 20–25 degrees) and 105 were definitely normal (CE greater than 25 degrees). The difference between these figures and those previously published by Muller and Seddon is due to my having accepted as definitely abnormal only those cases with a " CE " angle of less than 20 degrees, and as doubtful those with a " CE " angle from 20–25 degrees inclusive. Only 147 hips have adequate information for this investigation (Table VI). In twenty-nine, radiographs taken in infancy were available for study. Of these twenty-nine infants, seventeen

TABLE VII

UNFAVOURABLE FACTORS IN SEVEN CASES IN WHICH THE " HEALTHY " HIP REMAINED ABNORMAL AFTER CESSATION OF GROWTH

Number of cases	Plaster for less than six months	No plaster on " healthy " side	Treatment begun after five years of age
7	4	5	6

were found to be normal or doubtful and twelve abnormal when first examined. In assessing the normality or otherwise of these infant hips, Wiberg's method was found to be unsatisfactory because the centre of the femoral head could not always be identified with certainty and the dislocation on the other side made it difficult to establish the horizontal line. Accordingly Perkins's square was used to diagnose lateral displacement and Hilgenreiner's angle to measure the inclination of the acetabular roof to a line drawn between the triradiate cartilage of the two acetabula. This angle (the " H " angle) has been calculated to be 22·6 degrees in the normal infant at the age of one year and abnormal if greater than 25 degrees (Fig. 54).

When these criteria were applied twelve cases showed abnormal hip joints in infancy. Of these, six subsequently became normal, two improved and became doubtfully normal, and four remain frankly abnormal. Severin emphasised that these hips have a tendency to improve spontaneously, and this is supported by my finding only five definitely abnormal and two doubtfully normal hip joints on the healthy side among thirty-three adults with unilateral

TABLE VIII

ADULT CONDITION OF " HEALTHY " HIP RELATED TO AGE AT BEGINNING OF TREATMENT

Acetabulum CE angle in adult life	Number of cases	Average age at beginning of treatment
Less than 20 degrees	12	4·3 years
20–25 degrees	23	3·0 years
More than 25 degrees	71	3·1 years

dislocations which had not been treated. This is in contrast to the twelve abnormal " healthy " hips found among unilateral dislocations in twenty-nine cases radiographed in infancy.

The influence of treatment upon the development of these dysplastic hips must be considered. The supposedly normal hip was seldom mentioned in the case records and consequently there is uncertainty in most cases about the method and duration of treatment (if any) used on that side. In most cases, however, treatment was by double abduction hip spicas, and it is perhaps significant that eighty-one out of 105 patients in whom the CE angle exceeded 25 degrees in adult life received some form of treatment to the dislocated side. The notes are explicit in fifteen patients who had normal hips in adult life. Fourteen were

treated by a double hip spica throughout. In one a single spica was used for eighteen months from the age of five. The final result in this case, although normal (the CE angle measures 26 degrees), represents a deterioration from an earlier measurement of 33 degrees before treatment was started. In contrast, we find that of seventeen patients with abnormal hips when growth ceased (CE angle less than 20 degrees) five had no treatment whatsoever and

FIG. 48

FIG. 49

FIG. 50

FIG. 51

Evolution of dysplastic hip joint under treatment. Figure 48—At age of one year and six months. Right hip dislocated. Left hip subluxated. Acetabular roof very shallow. Figure 49—After one year in double abduction spica. Figure 50—At age of six. Acetabular roof now normal. Figure 51—Aged nineteen. Normal hip joint.

in five the method and duration are uncertain. In the remaining seven a definite adverse treatment factor (or factors) was present (Table VII).

The influence of the age at which treatment began upon the final result is shown in Table VIII, which includes 106 hips about which adequate information was available. The Table suggests that the age at which treatment begins influences the final result. Although the differences are small they are probably significant in that primary treatment was undertaken in all the cases between the ages of six months and seven years.

The evidence presented suggests that early diagnosis and vigorous treatment of an initially malformed hip joint may favourably influence its development just as they do in congenital dislocation of the hip. Figures 48 to 53 illustrate the application of these principles to the treatment of dysplastic hip joints in infancy and early childhood, with the consequent formation of a normal acetabulum. In contrast to these cases the hip joint illustrated in

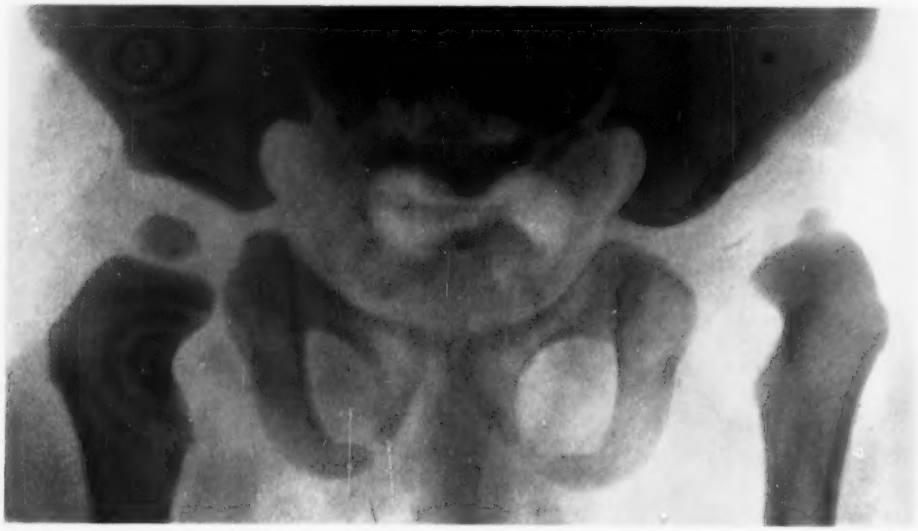

FIG. 52

Evolution of dysplastic hip joint under treatment. Figure 52.—At age of three. Left hip—congenital dislocation. Right femur lying away from acetabulum. The medial end of the upper femoral metaphysis has not yet crossed the ischial shadow. Figure 53.—Treatment by abduction plaster spicas on both legs for one year. In adult life hip is normal except for some valgus deformity of the neck. The "CE" angle is 25 degrees.

FIG. 53

Figures 54 to 57 was neglected, only the dislocated hip being placed in abduction. The final result is poor. This case is particularly interesting because the acetabulum on the side of the dislocation which was treated has developed better than its neglected though initially less abnormal fellow.

The above cases illustrate the possibilities that exist for curing this malformation and the price of neglect. Figures 58 to 60, however, illustrate the development of an abnormal

FIG. 54

FIG. 55

FIG. 56

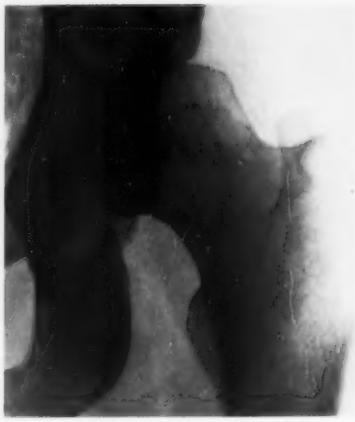

FIG. 57

Evolution of untreated dysplasia of hip. Figure 54—Aged four years. Congenital dislocation right hip. On the left the acetabular roof is shelving; the "H" angle is 35 degrees. Application of single abduction spica to right hip. Figure 55—At age of six. The right acetabulum has deepened considerably, but the left (untreated) remains shallow. Figure 56—In adult life. The right acetabulum is deeper than the left despite the original dislocation. Figure 57—The left acetabulum has remained shallow; the hip is subluxated.

acetabulum in a previously normal hip as a result of ill considered treatment on the dislocated side. This patient was allowed to walk in a unilateral hip spica in abduction for eight months. The other hip, which was inevitably adducted, failed to develop normally and at the early age of thirty an operation was necessary because of painful osteoarthritis.

FIG. 58

FIG. 59

FIG. 60

Evolution of dysplastic hip allowed to rest in adduction. Figure 58—Aged four and a half. Right hip—congenital dislocation. Left hip normal. Figure 59—One year later, after treatment by osteotomy and single abduction spica for dislocated hip. Walking was allowed in abduction. The left acetabulum, previously normal, is now shallow and does not cover the head of the adducted leg. Figure 60—At age thirty the previously normal hip is abnormal and shows a shallow acetabulum and butter-shaped head. Symptoms necessitated operation.

In conclusion, dysplasia of the hip is responsible for about one quarter of the cases of osteoarthritis seen in adults subjected to operation. A shallow acetabulum can be corrected or improved in youth by treatment in abduction. It may on the other hand be rendered worse or even produced if attention is not paid to a supposedly normal hip when this is

associated with a congenital dislocation on the other side. As judged by a small series of patients the earlier that treatment is begun the better the outlook for the acetabulum. The incidence of osteoarthritis is related to the degree of development of the acetabulum (Table IX). The average age of the patients was 31·5 years.

The results of this investigation into dysplasia of the hip joint serve only to support Putti's views published in 1933 and to re-emphasise the importance of early diagnosis and

TABLE IX

INCIDENCE OF OSTEOARTHRITIS RELATED TO DEVELOPMENT OF ACETABULUM

Adult acetabulum	Number of cases	Evidence of osteoarthritis				
		Radiographic only	Signs or symptoms only	Signs or symptoms + radiographic	Total cases	Per cent
CE less than 20 degrees	17	3	3	3	9	55
CE 20–25 degrees	25	0	2	2	4	16
CE more than 25 degrees	105	4	3	3	10	9·5

treatment in these patients if osteoarthritis is to be prevented in the future (Ponseti 1944, Ortolani 1948, Hart 1952). It is appropriate to end this section with a quotation from Putti's paper upon subluxation, " which is almost always overlooked and often neglected until it becomes obvious at an age when no radical cure is available and it becomes the cause of the syndrome of arthritis deformans—this should and could be corrected in infancy." Recognising that some might improve spontaneously, he continued: " If we have needlessly submitted a case of predislocation to a gentle and harmless form of treatment when it might have been lucky enough to reduce itself spontaneously, this is not in itself a ground for discrediting the method in which, according to our views, lies the only secret which can improve the results of treatment of congenital dislocation of the hip." (Putti used the term dislocation here because predislocation in his view might remain as a permanent subluxation or progress to a frank dislocation.)

THE INFLUENCE OF ISCHAEMIC CHANGES IN THE ETIOLOGY OF OSTEOARTHRITIS

Interruption of the blood supply to the femoral head causing massive necrosis is often followed by osteoarthritis and it is sometimes suggested that idiopathic disease is due to a lesser degree of vascular insufficiency causing cartilage degeneration insidiously (Wollenberg 1909, Watson-Jones 1942, Pridie 1951, Law 1952). Massive capital necrosis after a dislocation or transcervical fracture may be followed by osteoarthritis, and these cases are usually easily recognised. But osteoarthritis may also result from more subtle vascular occlusion in caisson disease (Phemister 1940), osteochondritis dissecans, and localised injuries to the femoral head (Figs. 61 to 64). In general, osteoarthritis is a disease whose incidence is greatest at an age when the peripheral circulation is decreasing. An association between arteriosclerosis and osteoarthritis has been claimed (Bezançon and Weil 1934, Monroe 1935), and Fletcher (1939) found that hypertension was present in 41·2 per cent of patients with osteoarthritis compared with a general incidence of 10·8 per cent among patients of comparable age.

The suggestion that osteoarthritis of unknown origin is due to progressive vascular insufficiency merits attention, for it is plausible, frequently advanced, and is the rationale of some forms of treatment such as the operations of forage (Graber-Duvernay 1932) and transposition of muscle flaps (Venable and Stuck 1946). The benefits that may follow treatment by short wave diathermy are believed to arise from an increase in the blood supply to the joint.

The radiographic appearances of advanced osteoarthritis may be readily confused with those of localised avascular necrosis. The familiar wedge-shaped density which appears below the area of greatest cartilage wear is not, however, due to infarction of this area but to vascular compact bone (Fig. 9). Cysts are not initially areas of necrosis but rather of vascular granulation tissue (Figs. 11 to 13). Similar changes occur on the acetabular side of

FIG. 61

FIG. 62

FIG. 63

FIG. 64

Serial radiographs showing progressive collapse of femoral head from avascular necrosis after fracture. Figures 61 and 62—Initial fracture seen in two planes. Figure 63—Three years after fracture. Figure 64—Six years after fracture. There is a resemblance between these radiological changes and those sometimes seen in idiopathic osteoarthritis.

the joint where the blood supply is profuse (Howe *et al.* 1950) and as protected from injury in its extra-osseous course as that to the femoral head is vulnerable.

When exposed at operation the femoral head may bleed very little if it is cut across after dislocation. This does not necessarily mean that it is avascular, for the torsion that accompanies forced lateral rotation may have obstructed the main capsular arterial supply. Anterior dislocation and elevation of the head may favour venous drainage through the marrow and necessarily divides the vessels within the ligamentum teres.

Although Walmsley (1915) believed that the arterial supply to the femoral head was reduced as age advances, Trueta and Harrison (1953) showed convincingly by angiography that a copious circulation and free intra-osseous communications persist in old age. Harrison *et al.* (1953), applying this method to a study of osteoarthritis, demonstrated an increased blood supply in the disease. My histological studies of thirty-five osteoarthritic femoral heads have constantly shown that the bone is well vascularised. Avascular necrosis was only seen when fragments of bone, undermined by cysts, broke away and were thus separated from their blood supply (Figs. 18 and 19).

It is of course possible that earlier ischaemic change had both caused the osteoarthritis and stimulated a vascular response in adjacent viable bone. This supposition cannot be supported, for dead bone undergoing revascularisation and substitution in situ was not seen. If this sequence were common, patients with severe peripheral occlusive vascular disease might be expected to show a high incidence of osteoarthritis in the limbs involved. But this is not so. Collins (1949) dissected knee joints amputated for arterio-sclerotic gangrene and found no increase in the incidence of joint degeneration. Kling (1938) made a similar clinical study and reached the same conclusion. I am indebted to Mr George Bonney for allowing me to study the case records of thirteen patients with occlusive vascular disease involving the great vessels distal to the aortic bifurcation and proximal to the division of the femoral artery into superficial and deep branches. All were over fifty years old, and in several an arterial block had been demonstrated by aortography, but in none was there radiographic or clinical evidence of osteoarthritis of the hip joint.

Degenerative joint changes similar to osteoarthritis have been produced experimentally by interruption of the blood supply to joints (Goldhaft *et al.* 1930, Miltner and Hu 1933). This reproduces in animals a well known happening in man but does not advance our knowledge of the etiology of most cases of osteoarthritis. Cecil (1930) suggested that synovial ischaemia might modify the quality of synovial fluid and so affect articular cartilage nutrition. The synovial membrane is, however, highly vascular in osteoarthritis and the synovial fluid does not appear to be modified except for some increase in the mucin content (Kling 1938). Pearson (1928) claimed that ischaemia of the spinal cord by interfering with vibration sense might render joints more vulnerable to minor injury and subsequent osteoarthritis. He could not, however, demonstrate a loss of the other forms of sensation transmitted by the posterior columns, and his views were not supported by pathological evidence.

In conclusion, and in company with Key (1930), Keefer *et al.* (1934), Harrison *et al.* (1953) and others, I found no evidence to support the view that osteoarthritis of unknown cause is often due to an ischaemic lesion in the femoral head. The notion that arterio-sclerosis is a cause of osteoarthritis was introduced in 1909 by Wollenberg, but in the same year Llewellyn Jones said of this association: " It must be admitted that any direct evidence of casual relationship if not entirely lacking is very scanty." The results of this investigation could well be summarised in those words.

OSTEOARTHRITIS OF UNKNOWN ORIGIN

This is the largest single group, numbering seventy-three (58·8 per cent) of the 124 cases analysed. The cause of osteoarthritis in this group has provoked many suppositions, of which some are " so meaningless as to be irrefutable " (Bennett *et al.* 1942). There is little to be gained by the perpetuation of hypotheses which affirm that a " sluggish bowel " (Ray 1932) or mycotic emboli (O'Reilly 1937), deranged purine metabolism (Gutman 1935) or " faulty elimination of metabolites " (Brailsford 1952) are factors of importance until methods are available by which such statements can be submitted to critical assessment. As an example of the more extravagant claims Ely (1922) suggested that the cysts were helminthic and that worms caused the disease. This was based on radiographic appearances alone. These have also caused confusion by their resemblance to those of ischaemic necrosis. Many of the hypotheses

have in common a tendency to ascribe all cases of osteoarthritis to one cause, whereas we already know that this disease is the ultimate stage of many seemingly dissociated joint disorders that have the ability to damage articular cartilage.

This study does not contribute significantly to the solution of this problem. Some observations seem, however, to be worth noting. Women were afflicted twice as often as men by both bilateral and unilateral disease. The age at onset of symptoms showed a peak for both sexes between the ages of fifty and fifty-five; so this is a disease of aging rather than old age. It was perhaps surprising to find that the average interval between the onset of symptoms and operation was only 4·9 years. This figure is based upon the practice of six different surgeons and indicates that deterioration is rapid in the more severe cases.

When the unilateral and bilateral cases are contrasted two distinguishing features emerge. The average age at which symptoms began was 52·5 years in the unilateral and 59·1 years in the bilateral. This may be significant, for the extremes of age incidence lie between relatively narrow limits. Secondly, there is a marked difference in the incidence of associated osteoarthritis in knees and spine between the two groups. This distinction is emphasised in Table X, which shows that bilateral disease is more often part of a polyarthritis than unilateral.

TABLE X

INCIDENCE OF ASSOCIATED OSTEOARTHRITIS IN OTHER JOINTS WITH IDIOPATHIC OSTEOARTHRITIS OF THE HIP

Type of disease in hip	Number of cases	Osteoarthritis in knee or spine	Osteoarthritis in knee and spine
Unilateral idiopathic	35	9	3
Bilateral idiopathic	36	21	15
Total	71	30	18

The knee or spine involvement was always severe enough to receive attention in deciding upon the method of treatment. The knee and spine are both functionally related to the hip joint, so their involvement might be regarded as secondary to the hip rather than coincidental. This is not supported by a similar study of dysplastic osteoarthritis (Table XI).

TABLE XI

INCIDENCE OF ASSOCIATED OSTEOARTHRITIS IN OTHER JOINTS WITH DYSPLASTIC OSTEOARTHRITIS OF THE HIP

Type of disease in hip	Number of cases	Osteoarthritis in knee or spine	Osteoarthritis in knee and spine
Unilateral dysplasia	16	4	0
Bilateral dysplasia	10	2	0
Total	26	6	0

The incidence here is low and probably not entirely accounted for by the earlier average age at operation (54·5 years compared with 59 years for idiopathic cases). An unstable hip on one side from birth would be expected to produce spinal osteoarthritis very commonly if the mechanical factor were dominant, whereas this happened on only three occasions among sixteen such patients and once in ten bilateral cases. Small though the numbers are, a similarity will also be observed between the incidence of multiple joint involvement in

unilateral idiopathic and unilateral dysplastic cases in contrast to the difference between these figures for the same types with bilateral disease.

Because of these differences it is suggested that unilateral and bilateral cases often represent the end-result of different causes. In the unilateral cases local factors may be more significant than the constitution..., thus perhaps accounting for the earlier onset of symptoms in monarticular arthritis which also characterises cases due to a long-standing mechanical fault. Minor degrees of dysplasia may cause some of these, for the average of onset in these patients is low (46·6 years); but this can seldom be recognised with certainty when osteoarthritis complicates the radiographic appearance. Although a similar criticism applies to the proposition of micro-trauma (Pommer 1920), ischium varum (Preiser 1907) and undetected adolescent coxa vara (Law 1952) as important contributory causes, the hypothesis that anatomical abnormality may be important in the etiology of these idiopathic cases is supported by the frequency with which anteversion is found at operation (Merle D'Aubigné and Postel 1954). Lastly, women are the victims twice as often as men of both unilateral idiopathic and dysplastic osteoarthritis.

The facts that bilateral osteoarthritis arises at a later age and is more often associated with polyarthritis tempt one to speculate that a constitutional upset contributes to the etiology in those cases not due to bilateral mechanical faults. The possible influence of the menopause (Hall 1938), and of pituitary dysfunction and acromegaly (Erdheim 1931), and of obesity (Fletcher 1939) have been assessed, and Silberberg (1936) have produced the disease experimentally by dietry and hormonal means. Haygarth (1805) first described the association in women of an osteoarthritic polyarthritis with Heberden's nodes. Kellgren and Moore (1952) confirmed and amplified this observation, believing that some unknown genetic factor which causes premature aging is responsible. Constitutional upsets such as these may well account for the disease in many cases of bilateral disease, which again is twice as common in women as in men.

In conclusion it must be admitted that little can be added as yet to McMurray's statement (1935) that "osteoarthritis represents the response of articular cartilage to many noxious agents of whose nature we know little." The commonest cause of articular cartilage injury is, however, advancing age (Bennett *et al.* 1942) and osteoarthritis is merely an acceleration and aggravation of this deterioration. A fuller understanding of normal cartilage metabolism will, it is believed, lead in the end to the discovery of the cause or causes of idiopathic osteoarthritis.

SUMMARY

1. The capsular changes in osteoarthritis of the hip and their pathogenesis are described, and it is concluded that symptoms are due mainly to this abnormality.

2. The clinical significance and pathogenesis of subchondral sclerosis, cysts, osteophytes, secondary subluxation and new bone formation on the lower border of the femoral neck are discussed.

3. These bony features which can be seen in the radiograph may, under certain circumstances, be correlated with the symptoms.

4. The influence of joint debris and capsular fibrosis upon the symptoms arising in other osteoarthritic joints is considered.

5. The mechanism by which osteoarthritis develops in hip joints with an anatomical abnormality is discussed in relation to the normal functional anatomy of the hip.

6. The evolution of osteoarthritis in dysplasia of the hip is considered with special reference to its diagnosis, prognosis and early treatment.

7. The supposition that osteoarthritis is commonly due to progressive ischaemia in the femoral head has been investigated and is rejected.

8. The cause of idiopathic osteoarthritis remains obscure but the evidence suggests that constitutional rather than local conditions in the joint account for many of these cases.

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SPONDYLOLISTHESIS*

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This paper is based on a review of forty-six patients with spondylolisthesis upon whom I have operated during the past five years. In all these cases a thorough exploration has been carried out in order to obtain a detailed picture of the anatomy of the lesion and in particular of the relations of the nerve roots. Armed with this information I have attempted to correlate the symptoms with the anatomical findings, and hence to provide a logical approach to the treatment of the condition.

DISCUSSION OF SYMPTOMATOLOGY

The symptoms of spondylolisthesis fall broadly into two groups, backache and pain referred to the lower limbs. The backache has usually and probably correctly been attributed to the instability of the affected segment, which increases the strains on the intervertebral ligaments and joints and ultimately causes severe localised osteoarthritis. From this aspect alone one might expect satisfactory cures from successful arthrodesis of the unstable and painful segment.

The cause of the radiating pain to the legs is a much more controversial subject and in recent years many suggestions have been put forward to explain its occurrence. These arise from two diverse schools of thought, the one which believes in the theory of referred pain, and the other which attributes symptoms to direct interference with the nerve itself. The first believe that pain from a local lesion in muscle, ligament or joint will be referred to the area of skin supplied by the same segmental nerve. The theory is attractive, but no one has succeeded in showing what this elusive local lesion may be, and, while the possible occurrence of referred sciatica must be kept in mind, it must also be realised that it may in fact be a myth. In favour of the theory that radiating pain is caused by direct interference with the nerve root, we have the indisputable fact that sciatica can be produced by compression of the root by a prolapsed intervertebral disc, or by rare but anatomically similar lesions such as a nerve root tumour, and that it can be relieved immediately and absolutely by removal of the lesion causing compression. It must now be accepted that many cases of sciatica are not due to prolapsed discs, but we are on sound ground when searching for other mechanical causes of root compression which might be expected to produce similar pain.

While running a special " back clinic " in the hospital service, I have had the opportunity of studying innumerable cases of sciatica. On clinical grounds these fall into two types which I have named—assuming for the time being that all are due to mechanical interference with the nerve root—the root compression syndrome and the root irritation syndrome. The first or compressive type presents marked physical signs with spasm of the lumbar muscles, deformity of the spine, a strongly positive straight leg raising test and possibly signs of nerve paralysis such as an absent ankle jerk, cutaneous hypoaesthesia or muscular weakness. These signs I believe indicate actual compression of the nerve root, the only common cause of which is a prolapsed disc. In the irritative type the distribution of pain is exactly the same, but physical signs are absent or slight. There is full mobility of the lumbar spine, the straight leg raising test is negative or only slightly positive, and neurological signs are absent. This syndrome I believe indicates nerve root irritation without actual compression. The commonest cause again is a prolapsed disc in which the size or position of the prolapse precludes severe compression of the root, and the acute disc prolapse which is making a symptomatic recovery will often pass from the compressive to the irritative type as a result

* Part I of an essay adjudged *proxime accessit* in the competition for the Robert Jones Prize and Medal, 1953.

of shrinkage of the prolapse. Nearly all cases of sciatica in which an exploratory operation reveals no disc prolapse belong to this root irritation syndrome and in many of them other possible causes of root irritation, such as the osteophytic edge of the vertebral body in the so-called "scarred disc," are apparent.

Cases of spondylolisthesis with radiating pain also fall into this category, clinical signs being almost invariably absent or slight. If the theory of root irritation is correct it should be possible to demonstrate mechanical interference with the nerve root as it passes through the spinal canal and out of the intervertebral foramen. My investigation of this series of cases has shown that such interference does in fact occur and offers a ready explanation of the symptomatology of the condition.

MORBID ANATOMY

Of the forty-six patients, thirty-eight had laminal defects, and eight had intact laminae Of the thirty-eight with defects, thirty-three had definite slipping (spondylolisthesis), and the remaining five had the typical defects in the pars interarticularis of the lamina but no slip (spondylolysis). The diagnosis of these latter cases is very easily missed unless routine oblique views are taken, and in one case I discovered the condition unexpectedly during an exploratory operation.

SPONDYLOLISTHESIS WITH LAMINAL DEFECTS

Of the thirty-three lesions, nineteen were at the lumbo-sacral level, eleven between L.4 and 5, and one between L.3 and 4. The remaining two patients had double lesions with L.4-5 spondylolisthesis and lumbo-sacral spondylolysis.

The lamina—During the exploration of these cases, the first striking feature observed is the looseness of the affected lamina. If the spinous process is held with forceps the lamina and its inferior facets can be rocked through quite a wide range, and this feature makes it easy to locate the correct level. In three cases, however, this laxity was absent, because of fibrous ankylosis of one or both of the apophysial joints. Apart from these three cases, the facets and the posterior joints appeared normal. In another three cases there was a complete spina bifida of the lamina which extended into the sacrum. It is important to remember this possibility, because damage to the cauda while stripping the muscles can easily be caused if it is not suspected.

The pseudarthrosis—It has been my policy to excise the loose lamina and its inferior facets completely, and this carries the dissection to the pseudarthrosis in the pars interarticularis of the lamina. It is noteworthy that the gap in the lamina is usually much smaller than the amount of slipping between the bodies of the vertebrae. The difference is due to a forward extension of the posterior part of the isthmus. I have not seen this fact referred to previously and it is obviously of considerable significance when considering the etiology of the condition.

The pseudarthrosis itself is an ill defined mass of fibrous tissue in which considerable mucoid degeneration has usually occurred. In these cases it has a crisp feel, almost like cartilage when cut with the nibbling forceps. This tissue merges into the neighbouring ligaments and I have never succeeded in defining its boundaries. It is often of considerable dimensions, bulging well beyond the bony margins of the pseudarthrosis and partly blocking the entrance to the intervertebral foramen. The emerging nerve root as it curves round the infero-medial border of the pedicle is an immediate anterior relation of this mass (Figs. 3 and 4), and in over half the cases in this series was bound to it by dense adhesions which could be separated only with difficulty.

The disc—When the lamina and the tissue within the pseudarthrosis have been cut back to the pedicles the step between the vertebral bodies is easily displayed by retraction of the dura. This step is covered with the annulus fibrosus of the deformed disc which presents a smooth glistening surface sloping gently upwards and forwards (Figs. 3 and 4). In one or two cases this disc tissue appeared a little fuller on one side than the other, but in none was

there anything resembling a disc protrusion. In many cases there was gross scarring of the disc with marked narrowing of the disc space, and thickened osteophytic edges extending backwards from the margins of both vertebrae (Fig. 6).

The dura—The dura makes a double bend as it descends between the lamina of the vertebra above and the posterior edge of the step below. It is always indented and often densely adherent at both these levels, particularly to the lamina and ligamentum flavum above. Even with severe deformities, however, these edges are not approximated sufficiently closely

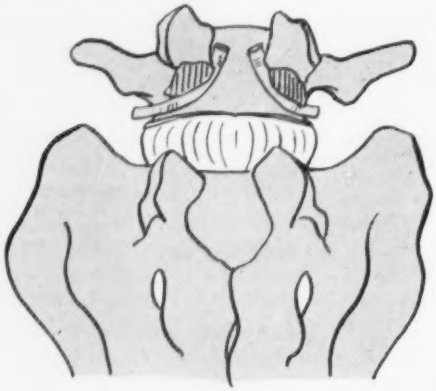

FIG. 1

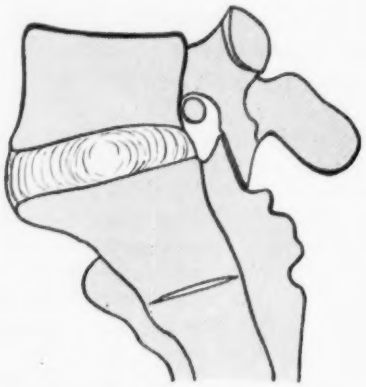

FIG. 2

Normal spine. Figure 1—Drawing from behind after total laminectomy, showing relations of L.5 root within the intervertebral foramen. Figure 2—Intervertebral foramen seen from within the spinal canal.

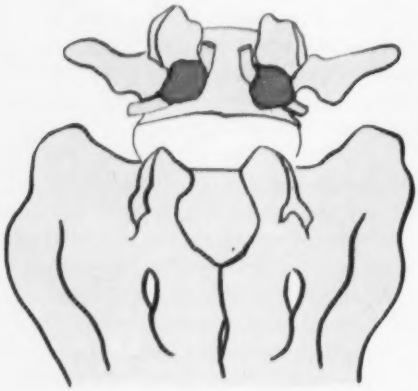

FIG. 3

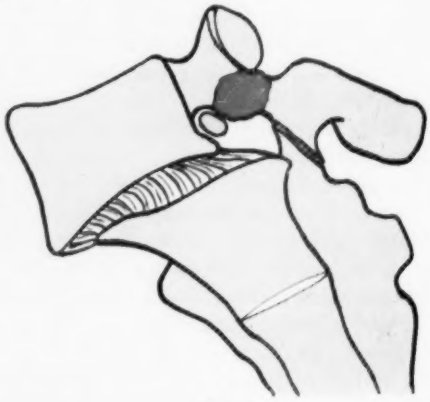

FIG. 4

Relations of nerve root in spondylolisthesis. Figure 3—Drawing from behind showing relations of nerve root to pseudarthrosis in pars interarticularis of lamina. Figure 4—Intervertebral foramen seen from within the spinal canal. Note intimate relationship of L.5 root to pseudarthrosis.

to cause actual compression of the cauda equina, although it may be tightly stretched across the edge of the step (Fig. 7).

The nerve root of the slipped segment retains its normal relationship to the pedicle and body of the forwardly displaced vertebra. The nerve root of the next segment below usually emerges from the dura just below the step, but in a few cases, and particularly in the more severe deformities, it arises just above it and passes downwards across its sharp posterior edge.

In front of the dura in the angle of the step is a " dead space " filled with fibro-fatty tissue and extra-dural vessels.

The intervertebral foramen—The intervertebral foramen receives scant attention in the anatomy books, and nowhere have I seen an accurate description of the relations of the nerve root as it passes through the foramen. These relations are of great importance when considering the causation of sciatica in spondylolisthesis and also in the scarred disc, and I propose to describe them in some detail.

In the normal spine the foramen may be divided into two distinct parts (Figs. 1 and 2). The upper half is bounded above by the pedicle, in front by the body of the vertebra and behind by the pars interarticularis of the lamina. It is demarcated from the lower half by the slightly prominent lower border of the vertebral body, and is occupied by the emerging nerve root. The lower half is bounded in front by the disc, below by pedicle, and behind by the apophysial joint. It is occupied only by fibro-fatty tissue and small vessels. The nerve enters the foramen by curving around the infero-medial border of the pedicle (Figs. 1 and 2), with which it is in close contact. It then proceeds directly laterally in a groove on the back of the vertebral body bounded above by the pedicle and base of the transverse process and below by the rounded lower margin of the body. At the extreme lateral border of the vertebra it turns downwards and forwards to enter the psoas muscle. Except at this extreme lateral

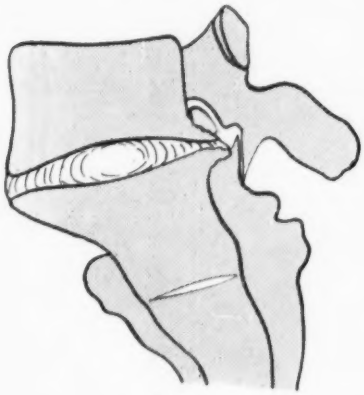

FIG. 5

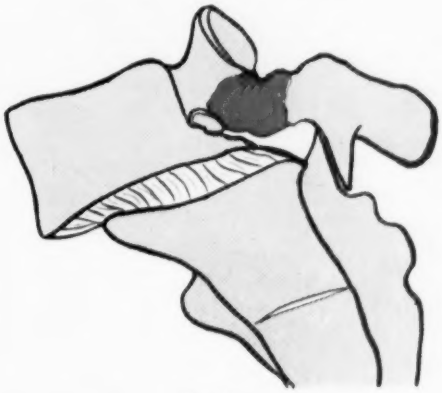

FIG. 6

Figure 5—Scarred disc. Intervertebral foramen seen from within the spinal canal. Note osteophytic ledge from L.5 body forming the floor of the neural groove. Figure 6—Spondylolisthesis with scarring of the disc. Note osteophytic ledge from L.5 body lying beneath the nerve root.

edge, the nerve is confined entirely to the upper half of the foramen and is never a direct relation of either the disc or the apophysial joint.

If the disc is scarred (Fig. 5), the backward projection of the osteophytic edge of the vertebra deepens the groove occupied by the nerve root, and if this edge is also thickened the groove will be constricted as well. The collapsed disc is projected backwards with the edges of the vertebrae and the lower half of the foramen may be thus almost obliterated, but this does not affect the root within the foramen.

In spondylolisthesis (Figs. 3 and 4) the foramen, although distorted, is actually enlarged. The nerve is always displaced forwards with the body of the slipped vertebra and occupies its usual position in the groove on the back of this body. The disc tissue attached to the edge of the vertebra now forms a sloping floor to this groove. The posterior wall of the upper part of the foramen is now occupied by the pseudarthrosis in the isthmus of the lamina, which frequently bulges into the foramen and impinges upon or adheres to the root. Although I have never seen the nerve actually constricted in this situation, the importance of this relationship cannot be over-emphasised.

If the spondylolisthesis is complicated by scarring of the disc (Fig. 6), the projecting

osteophytic ledge now forms the floor of the groove occupied by the nerve root. The thickened base of this ledge at its attachment to the body creeps upwards towards the pedicle and narrows this groove. Although this narrowing is seldom sufficient to constrict the root, in two cases definite and severe compression was demonstrated. In one case the canals between the lower surfaces of the pedicles and the osteophytic ledge were no more than one-sixteenth of an inch in width, and the roots traversing these canals were compressed into narrow bands. In the second case there was a similar appearance on one side, but on the other, where the canal was completely obliterated, the nerve had escaped by passing downwards within the spinal canal and then passing out directly laterally through the lower half of the foramen. Narrowing of the foramen in spondylolisthesis, and also in the scarred disc, has often been mentioned in the literature but without any clear appreciation of its

FIG. 7

Reconstructed radiograph showing relations of cauda equina. Cauda held forwards by lamina of L.4 and stretched across posterior edge of S.1 body.

mechanism. In this series the only demonstrable cause of such narrowing was the condition just described.

The extreme lateral part of the foramen is never easy to display at operation. In this situation the nerve turns downwards across the edge of the vertebral body and the lateral border of the disc to enter the psoas muscle or join the lumbo-sacral cord.

SPONDYLOLYSIS

Of the five cases of spondylolysis the lesion was at the lumbo-sacral level in three, and at the L.3-4 level in two. In addition, the two patients with double lesions both had lumbo-sacral spondylolysis. The appearance of the lamina and pseudarthrosis was exactly like that seen in cases of spondylolisthesis, and the nerve roots were adherent to the pseudarthrosis in three cases.

In one case there was a typical large globular disc protrusion on one side severely compressing the overlying nerve root. In all the other cases the disc appeared normal.

SPONDYLOLISTHESIS WITH INTACT LAMINA

Seven of the eight cases in this group conformed to a common pattern (MacNab 1950). All of these lesions were at the L.4-5 level and all in patients past middle life with considerable osteoarthritis of the spine.

On exploration the posterior intervertebral joints were found to be in a parasagittal plane instead of the normal oblique plane, and forward subluxation of the upper facets was apparent. The articular cartilage on the facets showed severe degenerative changes. The forward displacement of the lamina imposes an acute double bend on the dura and constricts the thecal canal between the lower edge of the lamina and the posterior edge of the step. But in none of these cases was this sufficient to cause a spinal block or actual compression of the cauda equina.

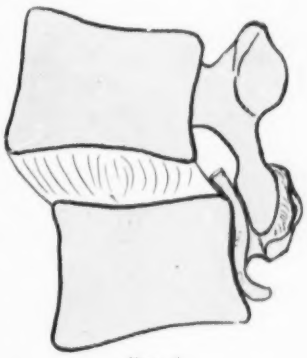

FIG. 8

Spondylolisthesis with intact lamina. Lateral view, showing the nerve root trapped between displaced facet and body of L.5.

The forward slip is limited by impingement of the displaced inferior facet against the body of the vertebra below (Figs. 8 and 9). In this situation the facet comes into close relationship with the nerve root passing towards the pedicle and intervertebral foramen of the segment below. In most of these cases the root had been squeezed medially and was densely adherent to the tip of the displaced facet.

The anatomy of the intervertebral foramen at the level of slip is like that of the other type of spondylolisthesis except for the important difference that the intact isthmus forms the posterior relation of the nerve root instead of the pseudarthrosis. The effects of scarring of the disc, which is usually marked in these cases, are the same.

The eighth case in this group falls into a different category. In this patient, a young woman of thirty, the lumbo-sacral joint was involved and slipping had occurred in consequence of developmental defects of the inferior facets of the displaced vertebra. The facet was completely absent on one side and represented by a small detached cartilaginous fragment on the other. The sacral facets were normal and this fragment was articulating with one of them. In the absence of any posterior support the lamina had slipped forwards and downwards until it impinged on the body of the sacrum and there trapped the cauda equina as it crossed the step (Fig. 10). This case is referred to in more detail later.

CAUSES OF SCIATICA

The clinical picture presented by cases of spondylolisthesis with radiating pain is nearly always that of the "root irritation syndrome," in which physical signs are absent or slight. It is for factors which might irritate the root rather than compress it, therefore, that we must search. During movements of the spine the nerve root glides within its bed and constriction or irregularities of this bed are likely sources of such irritation. The effect of such factors would be greatly increased by adhesions which fix the root at one point, so that it is undergoing constant traction.

In the foregoing description of the anatomical arrangements found in these cases, many possible causes of root irritation are apparent. I believe the most important are the laminal pseudarthrosis, the narrowing of the neural groove found in cases with scarring of the disc, and the forwardly displaced facets found in the cases without laminal defects.

The pseudarthrosis—The intimate relationship of the pseudarthrosis to the nerve root at its entrance to the foramen and the frequent fixation of the root to it by dense adhesions have already been described. I believe this is the most likely cause of radiating pain, and explains the frequent occurrence of such pain in cases of spondylolysis in which the other factors are not present. That movements occur at this false joint between the anterior and posterior elements of the vertebra has been clearly demonstrated by mobility films, and such movements will greatly increase the potential irritating properties of this joint. Alterations in the contents of the joint after strains and minor injuries might explain the frequency with which the symptoms are initiated by accidents. The pseudarthrosis affects only the nerve root at the level of slip—that is, the L.5 root in lumbo-sacral lesions and the L.4 root in L.4-5 lesions. In this series, as would be expected, the pain was of sciatic distribution in all lumbo-sacral cases, but was usually of femoral distribution with the higher lesions.

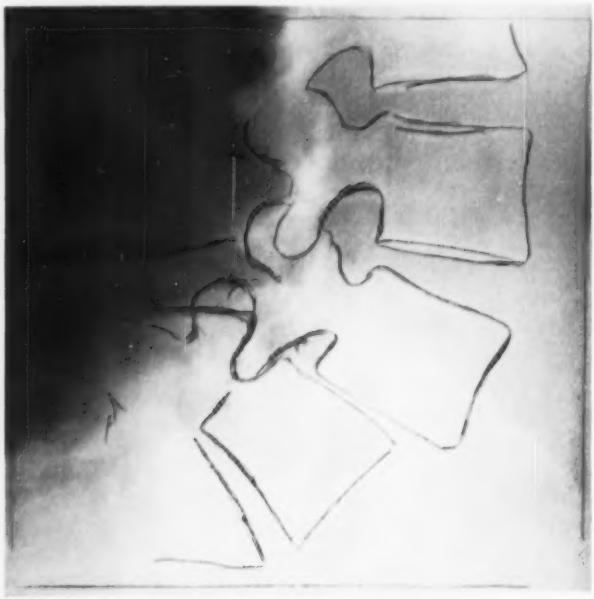

FIG. 9
Spondylolisthesis with intact lamina. Reconstructed radiograph showing inferior facet of L.4 impinging on body of L.5.

The neural groove—Although actual compression of the nerve root and its ganglion within this groove was demonstrated on only two occasions, in all cases with scarring of the disc the root was closely cushioned by the rugged ledge of the vertebral body, and I believe this to be another frequent source of root irritation. This feature is common to both types of spondylolisthesis, and also to uncomplicated cases of scarred discs.

Displaced facet—This factor is peculiar to the type of spondylolisthesis in which the apophysial joints are subluxated. The forwardly displaced facets come into intimate relationship with the nerve root descending across the step to enter the intervertebral foramen below (Fig. 8). The nerve may be trapped between the facet and the vertebral body, but is more commonly squeezed medially and becomes adherent to the tip of the facet. I believe that this is the commonest cause of radiating pain in this type of lesion and this view is supported by the distribution of pain, which was sciatic in all seven cases, although the slip was at the L.4-5 interval.

The satisfactory results after an operation that removes these sources of root irritation lend strong support to the view that they are the real cause of the radiating pain. Unfortunately, as all possible causes have to be dealt with in any particular patient, they cannot throw any light upon the question of which factor is responsible in any one case.

ASSOCIATED DISORDERS

Disc prolapse—In none of the cases of spondylolisthesis of this series was there anything resembling a prolapse of the affected disc. Indeed, if any considerable degree of slipping has occurred the disposition of the posterior part of the disc makes it extremely unlikely that such a prolapse would occur. Even if the nucleus succeeded in escaping posteriorly it would presumably bulge into the " dead space " between the step and the dura and would be unlikely to cause root compression.

In one case of spondylolysis a typical large well defined protrusion had occurred and was undoubtedly the cause of the patient's sciatica. This case presented the clinical picture of " root compression " with marked physical signs, and a tentative diagnosis of disc prolapse was made before operation. It seems likely that the local instability associated with spondylolysis, and possibly also with slight degrees of spondylolisthesis, might well predispose to prolapse of the nucleus at this level, and the possibility of its presence should always be borne in mind.

In two other cases of spondylolisthesis with laminal defects a typical protrusion had occurred at a disc above or below the level of slip. In one case the spondylolisthesis was lumbo-sacral and the protrusion at the L.4-5 disc, and in the other this picture was reversed. In both of these cases the condition was suspected clinically before operation. It is possible, however, that a disc prolapse at an adjacent level might give rise to a " root irritation " picture without physical signs, and be indistinguishable from an uncomplicated case of spondylolisthesis. I believe therefore that in all cases with sciatic pain exploration of the adjacent disc should never be omitted.

Tuberculosis—I have seen only one reference in the literature to the association of spondylolisthesis with tuberculosis of the spine. Nissen-Lie (1941) reviewed the spinal radiographs of a large number of patients admitted to one hospital, and among 267 who had spinal tuberculosis he discovered five cases of spondylolisthesis. In three cases the two conditions affected the same vertebrae. Tuberculosis occurred in two cases of my series and in one case involved only the deformed segment.

Case 1—This patient was under conservative treatment for severe sciatica for some months before operation was performed. Radiographs showed a moderate degree of lumbo-sacral spondylolisthesis, but no evidence of an infective lesion in the spine or pelvis. Marked lumbar rigidity with a sciatic scoliosis was present, and it was thought that this was probably due to a coincidental prolapsed disc. At the operation, which included a thorough exploration and curettage of the disc, nothing unusual was detected and some bone chips from the ilium were used in the arthrodesis. The wound healed normally, but some weeks later, while the patient was still being nursed in a plaster bed, a sinus developed which was eventually proved to be tuberculous. Although the infection spread to involve the lumbo-sacral joint, a review of the pre-operative and later films shows the probability of an original infection in a sacro-iliac joint which may well have been responsible for the symptoms which brought him to hospital.

Case 2—This patient complained only of backache, had only slight muscular rigidity, and was found to have a L.4–5 spondylolisthesis. The radiographic appearance of the affected disc led to a discussion of the possibility of an infective lesion, but this diagnosis was thought to be so improbable that an exploratory operation was finally advised. During this exploration nothing abnormal was seen until the disc was incised, when tuberculous pus exuded. The disc was gently curetted and nothing further was done. The patient has made satisfactory progress after treatment along more orthodox lines.

The occurrence of two cases of tuberculosis in this series of forty-six cases of spondylolisthesis may be a chance coincidence. It is a sharp reminder, however, that many

conditions can cause low backache and sciatica, and that the radiographic demonstration of an obvious lesion like spondylolisthesis does not necessarily settle the diagnosis.

Cauda equina lesion—Many authorities have denied that a cauda equina lesion can be produced by spondylolisthesis, but at least three cases have been reported in recent years (Hulbert 1948, Arden 1949, Turney 1952). Turney's case had bladder dysfunction and saddle anaesthesia and one of my cases presented a similar picture. This was the atypical case of spondylolisthesis with intact lamina in which slipping had occurred as a result of congenital deficiencies in the inferior facets.

Case 3—The patient, a woman of thirty, had never had any serious backache and no sciatica, but complained of stress incontinence for the previous two and a half years. On examination she had typical saddle anaesthesia involving the inner sides of the thighs and the anal and vulval skin. Evacuation of the bladder could be effected normally and the incontinence appeared to be

FIG. 10

Case 3—Lumbo-sacral spondylolisthesis with intact lamina but absent inferior facets. Reconstructed radiograph showing the cauda equina trapped between the lamina of L.5 and the posterior edge of the sacrum.

due to failure of the usual reflex closing of the sphincters during sudden stresses, as a result of the anaesthesia. Radiographs showed slipping to the extent of half the diameter of the vertebral body, and a myelogram demonstrated a complete block at the lower border of the displaced lamina. At operation the lamina was found to be resting against the posterior edge of the step and the cauda equina was tightly compressed into a thin band as it passed between (Fig. 10). Decompression was performed by removing the lamina and the prominent angle of the sacral body, but no recovery has occurred in the two and a half years since the operation.

It is of some interest that this patient's brother has also been under my care with lumbo-sacral spondylolisthesis. His disability is slight and it seems unlikely that I will have an opportunity of exploring his back. Radiographs show that the typical pseudarthroses in the isthmus are almost certainly present.

This case is exceptional in that the complete absence of facets had allowed the lamina to slip farther forward than usual and also to descend more nearly to the body of the sacrum. Intact facets would either impinge on the back of the body and prevent further slipping,

or if they passed on to the upper surface of the body would elevate the lamina and so prevent severe damage to the cauda equina. It is probably true that the usual type of spondylolisthesis with defects in the isthmus, in which the lamina is left safely behind, could never produce a cauda equina lesion.

ETIOLOGY

The findings in this series of cases do not throw much light on the origin of the defects within the isthmus, although a few observations are of significance. In several cases the laminal defect was much narrower than the step between the vertebral bodies, and this was brought about by an elongation of the posterior part of the isthmus. In these cases, therefore, both the defects and the slipping must have been present at a very early stage of skeletal development. That the lesion is the result of developmental defects is also supported by the

FIG. 11

FIG. 12

Case 4—Lumbo-sacral spondylolysis. Figure 11—Initial radiograph. Laminal defects are visible but they were not detected when the patient first attended. Figure 12—Two years after first attendance. Spondylolisthesis has now occurred.

occurrence of double lesions in two cases, and by its association with the other developmental defect of spina bifida in three cases.

When defects are present, however, the actual deformity caused by slipping can certainly be acquired. This is well illustrated by one of my cases (Figs. 11 and 12), in which there was a transition from a simple spondylolysis to a well marked spondylolisthesis while the patient was under my care.

Case 4—This patient, a woman of fifty-one, attended hospital complaining of chronic low back pain. Although the routine radiographs (Fig. 11) show laminal defects to be present these were not noticed at the time. She improved after a course of physiotherapy, but returned a year later when further radiographs (Fig. 12) showed well marked spondylolisthesis, which has since been stabilised by arthrodesis.

In this case there was no history of injury and the transition occurred, presumably gradually, without any dramatic change in the symptoms. It is possible, however, that

such a change could be brought about by an accident, and this point is of great importance from a medico-legal point of view. Cases of spondylolysis are easily missed unless oblique views are taken, and I believe it is dangerous to make a final assessment of any back problem without them.

I have not included in this series any cases of traumatic spondylolisthesis. The few cases I have treated have all resulted from fractures through the facets or through the main part of the lamina, and symmetrical fractures through the isthmuses must be a rarity. In all my cases open reduction has been effected without difficulty. The differentiation of traumatic from congenital cases is not always easy, as is illustrated by the following case.

Case 5—This patient was brought to hospital after falling twenty feet from a roof, and complained of severe low back pain. Radiographs showed a lumbo-sacral spondylolisthesis which was believed to be traumatic, and operation was performed a few days after admission. The typical findings of a long-standing deformity were present, and there was no evidence at all of recent injury, such as haematoma or ruptured ligaments.

The initiation of symptoms by injury in a lesion which had previously been symptomless is a well recognised phenomenon, and this again can raise difficult medico-legal problems.

TREATMENT

Once the detailed anatomy of this condition is understood and the incidence of the commoner associated conditions is appreciated, a sound operative procedure for its treatment becomes possible. This may be divided into three stages—exploration, decompression of the nerve roots, and arthrodesis.

EXPLORATION

Adequate exposure of the deformed segment requires complete removal of the loose lamina and its attached inferior facets together with the ligamentum flavum above and below. If there are no laminal defects the lamina should be removed laterally as far as its junction with the pedicles.

The incidence of disc prolapse at an adjacent segment is sufficiently common to make exploration of these segments imperative in all cases. If this opportunity of doing so is lost and sciatic pain persists after the operation, the future conduct of the case becomes a very grave problem indeed. If the pain is of sciatic distribution the exploration may be confined to the lowest two disc spaces, unless an abnormal number of lumbar vertebrae and the possibility of a prefixed plexus make a wider exploration advisable. In lumbo-sacral lesions, the lumbar 4-5 disc is easily displayed after nibbling away the lower edge of the fourth lamina. If the fourth lumbar vertebra has slipped, a hemilaminectomy of L.5 will be necessary to explore the lumbo-sacral disc. If the pain is of femoral distribution, suggesting interference with the fourth lumbar nerve root, the L.3-4 disc should be explored also.

A really adequate exploration is only possible in the absence of severe haemorrhage. Bleeding not only obscures the details but makes the operation much more lengthy and dangerous. We have found that preliminary spinal anaesthesia diminishes the bleeding to negligible proportions so that the operation can be performed both efficiently and quickly without serious deterioration in the patient's condition.

DECOMPRESSION

In the cases of spondylolisthesis with intact laminae excision of the lamina and inferior facets will remove the main cause of root irritation. In both types of spondylolisthesis the cauda equina and the root passing to the intervertebral foramen below may be stretched over the posterior edge of the step and in these cases this edge must be removed with a gouge (Fig. 13). This is particularly important when there is a sharp osteophytic edge on the lower vertebra.

When the lamina has been removed the pseudarthrosis itself is dealt with after any

adhesions between it and the nerve root have been separated. All the tissue filling in the defect must be nibbled away. The anterior part of the isthmus must then be removed until the root is seen to be lying quite freely as it curves round the infero-medial border of the pedicle.

Finally any constriction of the groove occupied by the nerve root on the back of the vertebral body must be released. This is done by removing the osteophytic ledge of the vertebra with a gouge right out to its extreme lateral border (Fig. 13). This procedure necessitates cutting into the disc tissue filling in the step, and it is then essential to resect this tissue and curette the disc space.

The principle of decompressing the nerve roots by foramenotomy was described by Briggs and Keats (1947) who stressed the importance of removing the facets of the apophysial joints. The facets themselves lie below and posterior to the roots and are never an immediate relation of them; as the medial facet has already been removed, however, there is no advantage in preserving the lateral facet, and its removal makes it easier to deal with the osteophytic ledge. It cannot be too strongly emphasised, however, that the whole purpose of foramenotomy is defeated unless the pseudarthrosis and this osteophytic ledge are adequately removed.

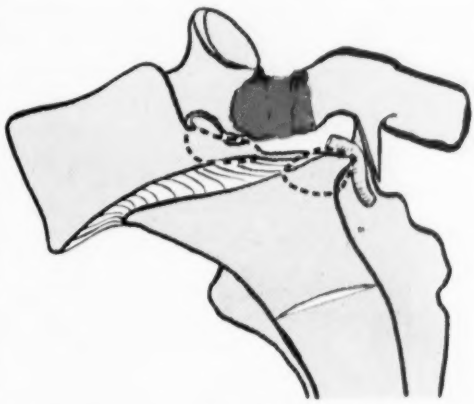

FIG. 13

Spondylolisthesis. To effect decompression of both L.5 and S.1 roots, the areas of bone outlined by interrupted lines must be removed.

Results—In this series of cases the satisfactory results from this treatment of the nerve root are strong evidence that the cause has been removed. Before operation all but three of the patients complained of leg pain and in every case the pain disappeared after the operation. Although the lengthy period of post-operative immobilisation may have contributed to this relief, in several of them severe and intractable sciatica had persisted in spite of treatment in a plaster corset. The relief of their pain after the operation was sufficiently dramatic to make it almost certain that the cause of it had been removed. This was particularly striking in three elderly patients whose sciatica persisted in spite of prolonged conservative treatment in plaster corsets. On account of their age a simple decompression without any attempt at fusion was performed and they were allowed up in a Jordan's brace four weeks after the operation. Relief of pain was complete and immediate in all three cases. In one case, however, further slipping has occurred (Figs. 14 and 15). Although this has not progressed and has not caused symptoms this procedure is obviously potentially dangerous and I would not feel justified in repeating it.

The follow-up period of the cases in this series varies between six months and five years. About one-fifth of the patients have had some recurrence of their leg pain which in most cases has been only temporary, and, in some, of different distribution from the original pain. It is only possible to guess at the causes of these recurrences. They could be due to inadequate freeing of the nerve root by the decompression operation, or to new bone formation from the cut surfaces. I think a more likely explanation is that the nerve root became involved in the dense fibrosis which so often follows any extensive operation within the spinal canal. This itself can cause root irritation from the constant traction produced by spinal movements, and this will not, of course, be affected by a local arthrodesis of the spine.

Posterior rhizotomy—In view of these recurrences I now believe that, whenever possible, the operation should be completed by division of the sensory nerve root. I have only recently

adopted this procedure as part of my routine treatment, and cannot yet say whether or not this will improve the results. Division of one root appears to be safe, but it is dangerous to divide two roots on the same side because anaesthetic patches on the foot and a trophic ulcer may be the result. I have seen one such case, operated upon elsewhere, in which we had to amputate the great toe. The method must be restricted, therefore, to cases in which it is apparent which root is affected, but in most cases this is not difficult to decide.

ARTHRODESIS

Successful arthrodesis of the unstable segment is essential if all back disability is to be relieved. In all cases in this series in which sound bony fusion can be demonstrated the results are excellent. In many cases the bone grafts have not become incorporated but firm

FIG. 14

FIG. 15

Case 6—Lumbar 4 5 spondylolisthesis with intact lamina. Figure 14—After operative decompression without arthrodesis. Figure 15—One year later. Considerable further slipping has occurred.

fibrous ankylosis has nevertheless been attained. The results of these cases are nearly but not quite as good as those with bony fusion. In all cases in which demonstrable instability remains there has been some persistent back disability which has prevented a return to heavy work and in most cases has been a source of considerable discomfort.

The question of arthrodesis of the lumbo-sacral region of the spine, especially after laminectomy, poses many problems and I wish to deal with these in a separate paper. I believe that the usual methods in which a long posterior bone graft is used to bridge the unstable segment are thoroughly unsatisfactory. Cleveland, Bosworth and Thompson (1948) quoted a pseudarthrosis rate of 35 per cent and I suspect that it may be even higher. The ideal procedure would employ a short intersegmental graft crossing only the unstable joint. After laminectomy such a fusion can only be effected between the vertebral bodies or between the lateral elements of the vertebrae—the pedicles or transverse processes. My results with

FIG. 16

Case 6—Lumbo-sacral spondylolisthesis treated by bilateral tibial ala-transverse grafts. Six months after operation bone grafts are incorporated and buttressed by new bone.

FIG. 17

Case 8—Lumbar 4–5 spondylolisthesis and lumbo-sacral spondylolysis. Separate inter-transverse grafts L.4–5 and L.5 ala. Six months after operation grafts are well incorporated.

FIG. 18

Case 9—Lumbar 4–5 spondylolisthesis, eight months after bilateral inter-transverse fusion with tibial grafts. Note massive buttressing at inner side of graft on left side.

grafts inserted between the bodies from behind have shown that such grafts seldom if ever become incorporated, although firm fibrous ankylosis is the usual result. I have no experience of the anterior methods of fusion, but apart from their danger they have the grave disadvantage that they cannot be combined with decompression of the nerve roots, which is essential in all cases with sciatica.

I have had many beautiful results from inter-transverse or ala-transverse grafts (Figs. 16 to 18) and I believe that the answer to this problem lies in the perfection of this technique.

SUMMARY

1. A detailed analysis of the anatomy of spondylolisthesis reveals many causes of serious interference with the nerve roots.

2. These anatomical findings can be correlated with the symptoms.

3. In association with spondylolisthesis, cases of disc prolapse, tuberculosis, and a cauda equina lesion are described.

4. Adequate decompression of the affected nerve root is essential in all cases with sciatica. The operative procedure is described.

5. The problem of arthrodesis will be discussed fully in a separate paper.

I wish to express my gratitude to Mr R. G. Pulvertaft for his help and encouragement during the progress of this work, to Dr G. Q. Chance for his constant assistance with the radiological aspects of the work, and to Dr K. P. Veliskakis for the drawings.

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MUSCLE RECOVERY IN POLIOMYELITIS

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How much recovery will take place? For how long will improvement continue? In every case of paralytic poliomyelitis these two questions are asked. Some would echo Courtney (1896) in answering that "the disease is so variable that it is very difficult to say anything about prognosis at all." While this may be the correct answer during the acute stage of the disease, it should be possible, after the first four weeks, to give a less guarded reply.

With the development of manual methods of muscle testing first introduced by Lovett and described by his assistant Wright in 1912, the grade of paralysis of a muscle may be defined in terms that allow the progress of its recovery to be followed. In recent years results obtained by these methods have been analysed to show the extent of muscle recovery in poliomyelitis (Harry 1938, Carroll 1942, Lenhard 1943, Watkins 1949, Green 1949, Grossiord and Husson 1953). Though these studies indicate the general potentialities for recovery in paralysed muscles, their usefulness in clinical practice is limited. In most, the muscles investigated have been too few and the tests too infrequent to allow the progress of muscle recovery to be followed in detail. The recovery of individual muscles has received attention from only one author (Skinhoj 1949) and differences that may exist in the rate of recovery at different ages have not been investigated.

The object of this paper is to show how a knowledge of muscle recovery may be of practical value in the management of patients in the convalescent stage of poliomyelitis.

MATERIAL AND METHOD

Between November 1949 and December 1950, 185 patients who had suffered a recent attack of paralytic acute anterior poliomyelitis were admitted to the Stanmore section of the Royal National Orthopaedic Hospital. In 149 patients the onset of the paralysis had occurred less than thirty-two days before admission. These were the cases in which a three-year study of recovery was begun.

Each patient was examined on ten occasions, at one, two, four, six, eight, ten, twelve, eighteen, twenty-four and thirty-six months after the onset of the paralysis. A complete muscle test was made on each occasion. So far as possible, testing was always performed with the patient in a warm room, after rest. Standard methods of manual muscle testing were used (Daniels, Williams and Worthingham 1947; Kendall and Kendall 1949). The grade recorded depended upon the power of a muscle in the third of three contractions through the greatest range of movement of the joint concerned. The scale of muscle power ranging from 0 to 5 recommended by the Medical Research Council (1942) was used during the making of the observations, though, for reasons given below, it was modified in the analysis of results. All the tests were made by the author.

At the end of three years a complete follow-up had been obtained in 142 patients, seven of the original number having moved to a distance too great to justify repeated attendances for examination.

All the patients received the same treatment during the investigation. This was the standard treatment given at the hospital and consisted in daily passive movements to the joints and active exercises to the muscles of an affected limb up to, but not beyond, the point of fatigue. After three to four months, increasing functional activity was added to the regime in most cases, with diminishing attention to individual muscles. The duration of in-patient treatment varied between one month in cases of mild paralysis of one upper limb,

and two years or more in severely paralysed individuals. The mean duration of active physiotherapy was ten months.

The accuracy of manual muscle testing—The investigation of muscle recovery by manual methods has recently been the subject of some criticism by those who advocate a return to mechanical methods of measurement of muscle power (Russell 1952)—notably that, in the assessment of the power of a muscle, it is not certain that two observers will assign the same grade to it.

In most muscles and muscle groups the Medical Research Council scale can be used to give an unequivocal result. An incorrect assessment is almost always due to ignorance of the method, inattention to detail, lack of appreciation of the action of the muscle under test, or the deception of the trick action of another muscle. A high degree of objectivity is necessary in manual muscle testing; all too frequently the task is delegated to the physiotherapist who is treating the patient, and the desire to record an increase in power may supersede the strict evidence of the test.

Ideally, therefore, muscle tests should be made by someone who does not see and treat the patient every day. They should be performed without reference to the results of previous tests and the same observer should be responsible for all the tests in a given patient. It is true that the assessment of the power of certain muscles, such as the muscles that act upon

TABLE I

THE DISTRIBUTION OF PARALYSIS IN MUSCLES OF THE LOWER LIMB

Muscle	Total of all grades of paralysis one month after the onset of the paralysis	Number completely paralysed three years after the onset of the paralysis
Hip flexors	198	34
Hip abductors (gluteus medius and minimus)	201	47
Hip adductors	193	36
Gluteus maximus	203	33
Biceps femoris	202	54
Inner hamstring muscles	204	43
Quadriceps	204	46
Tensor fasciae latae	189	38
Tibialis anterior	182	103
Tibialis posterior	169	89
Peronei	165	60
Extensor hallucis longus	157	66
Extensor digitorum longus	161	63
Triceps surae	162	57
Flexor hallucis longus	154	84
Flexor digitorum longus	155	85
Intrinsic muscles of the foot	140	24

the digits, is difficult to make in the exact terms of the Medical Research Council scale and is partly dependent on the pure judgement of the particular observer; even so, the assessments of that observer should be consistent.

In this investigation I have checked the constancy of my own muscle testing by occasionally repeating a test after an interval too short for any recovery to have taken place, so that the results of the second test should have been identical with those of the first. The muscles listed in Tables I and II, and included in the analysis to follow, gave constant results. The results for some muscles, such as the small rotator muscles of the hip, serratus anterior, and the muscles of the trunk, proved to be unreliable and were therefore excluded from the analysis. On rare occasions the results of muscle tests could be compared with the findings in muscle at necropsy. The proportion of residual normal muscle fibres always corresponded well with the clinical assessment in the muscles listed in Tables I and II. In muscles that were unreliable in the clinical test the clinical grade was often at variance with the necropsy findings.

An enormous mass of data resulted from the observations, there being over 110,000 individual muscle assessments. At the first examination there were 3,033 muscles in 211 lower limbs and 1,905 muscles in 126 upper limbs whose power had been graded as less than normal. It is the progress of recovery in these muscles that will now be described.

The uniformity of the Medical Research Council scale of muscle power—Because the power of a muscle is denoted by a numeral it is tempting to imagine that the figures bear a mathematical relationship to one another. Although, from the arbitrary nature of the system of grading, any such relationship would be coincidental, the clinical impression gained from experience in muscle testing is that some uniformity does exist in the "steps" between one grade and the next: that, for instance, the amount of recovery indicated when a muscle increases in power from grade 2 to grade 3 is comparable with that when it increases from grade 3 to grade 4. The data from this study have been used to deduce mathematically whether this is the case.

TABLE II

THE DISTRIBUTION OF PARALYSIS IN MUSCLES OF THE UPPER LIMB

Muscle	Total of all grades of paralysis one month after the onset of the paralysis	Number completely paralysed three years after the onset of the paralysis
Trapezius	81	3
Pectoralis major	108	9
Deltoid	127	25
Lateral rotators of shoulder	107	14
Latissimus dorsi	95	11
Triceps brachii	118	15
Elbow flexors	97	10
Extensor carpi radialis longus	69	17
Extensor carpi radialis brevis	67	8
Extensor carpi ulnaris	65	7
Extensor digitorum	66	8
Extensor pollicis brevis	65	10
Abductor pollicis longus	65	12
Extensor pollicis longus	68	5
Pronators	68	11
Flexor carpi ulnaris	62	12
Flexor carpi radialis	68	16
Flexor pollicis longus	58	8
Flexor digitorum profundus	56	2
Flexor digitorum sublimis	56	5
Opponens pollicis	75	30
Flexor pollicis brevis	66	23
Abductor pollicis brevis	75	30
Interossei	63	13
Hypothenar muscles	60	16

The proportions of muscles in grade 1 that reached grade 2, of grade 2 muscles that reached grade 3, of grade 3 muscles that reached grade 4, and of grade 4 muscles that reached grade 5 in the interval between the first and second muscle test (one to two month interval) were calculated. Proportions were worked out in the same way for the two to four month, four to six month, and six to eight month intervals. During any of these intervals, the proportion of grade 4 muscles that increased in grade was consistently about one-half that of any of the other grades; that is to say, while it was as easy for a muscle to proceed from grade 1 to grade 2 as from grade 2 to grade 3 or as from grade 3 to grade 4, it was nearly twice as great a step for it to proceed from grade 4 to grade 5.

A second analysis was then made in which muscles that had been assigned a grade of 4+ were considered as being in a separate grade; the possible moves in grade that could occur were then: 1 to 2, 2 to 3, 3 to 4, 4 to 4+, 4+ to 5. The results using this modified scale showed that a surprising uniformity now existed in the steps between all the grades.

As a result of this finding, all muscles previously designated grade 4+ were rechristened grade 5, and all those of grade 5 became grade 6. The new 0 to 6 scale, in the terms of which subsequent analyses were made, becomes:

0 = No contraction.
1 = Flicker or trace of contraction.
2 = Active movement with gravity eliminated.
3 = Active movement against gravity.
4 = Active movement against gravity and some resistance.
5 = Active movement against gravity and considerable resistance.
6 = Normal power (within the limits of manual assessment).

THE RATE OF RECOVERY IN THE MUSCLES OF THE LOWER LIMB

Of the 3,033 lower limb muscles of all grades of paralysis at the first muscle test 962 remained completely paralysed (grade 0) throughout the three-year period. These irrecoverable muscles never partook in the process of recovery and were, therefore, separated from the total. Evidence from muscle biopsy and necropsy suggests that permanently paralysed muscles are

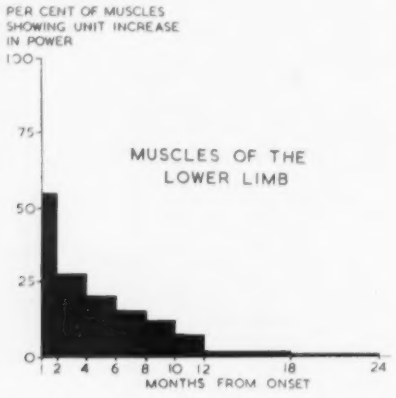

FIG. 1
The rate of recovery in the muscles of the lower limb.

completely denervated at the acute stage of the disease, and findings in the anterior horn of the spinal cord support this view (Sharrard 1953). The remaining 2,071 muscles form a "working total" in which recovery during the one to two month interval may be calculated.

The unit of recovery in this analysis is an upward move in grade. An upward move of one grade, as from grade 2 to grade 3, represents one unit of recovery; an upward move of two grades as from grade 2 to grade 4, represents two units of recovery. The total number of such moves in grade between the one month and two month muscle test was found to be 1,128; that is, 54·5 per cent of the working total of 2,071 recoverable muscles.

At the two month muscle test sixty-one muscles had reached grade 6 and, so far as muscle testing was concerned, had become incapable of further recovery. This number was subtracted from the original total of 2,071 muscles to give a *new* working total of 2,010 muscles for the calculation of recovery in the two to four month interval. The total number of upward moves in grade during this period was 1,114, that is 55·4 per cent of the working total of 2,010 muscles. But this proportion of muscles recovered during a period of two months, so that the rate of recovery per month becomes 27·7 per cent.

The rate of recovery at subsequent intervals was calculated in the same way; the results are shown in histogram form in Figure 1. From a high initial figure the rate of recovery

falls rapidly at first and then more gradually until, at the twelfth month, less than 5 per cent of muscles move in grade each month. At this time, fourteen-fifteenths of the total possible recovery has taken place.

No increase in grade was observed after the twenty-fourth month except in rare instances. Thus after the correction of a position of deformity at a joint a muscle that was previously working at a mechanical disadvantage or had been subject to overstretching was able to increase its activity and power. It must be emphasised, though, that twenty-four months

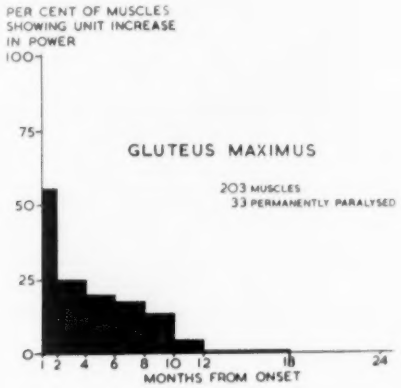

FIG. 2

The rate of recovery in the gluteus maximus.

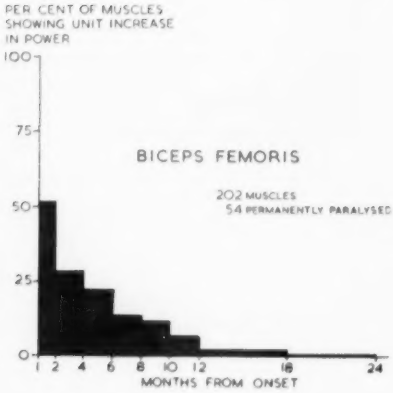

FIG. 3

The rate of recovery in the biceps femoris.

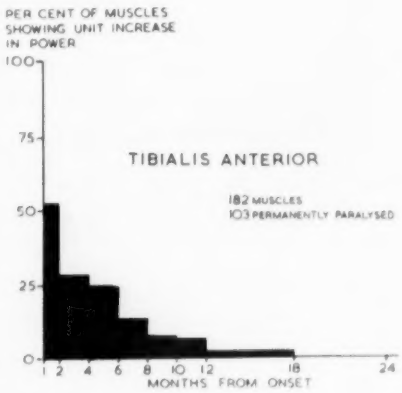

FIG. 4

The rate of recovery in the tibialis anterior.

does not necessarily mark the end of *functional* recovery, to which no final date can be given in any one patient.

The same method of calculation was used to discover the rate of recovery in the seventeen individual muscles of the lower limb. Figures 2, 3 and 4 show the results found for gluteus maximus, biceps femoris and tibialis anterior. The interesting and unexpected finding is that all the individual muscles of the lower limb show almost identical rates of recovery, both in the shape of their curve and in their absolute data. The impression gained from clinical observation had been that the rate of recovery differed in individual muscles. For instance,

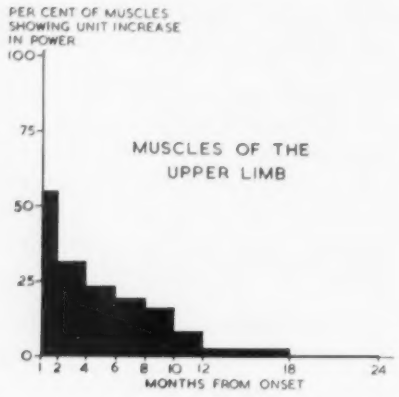

FIG. 5
The rate of recovery in the muscles of the upper limb.

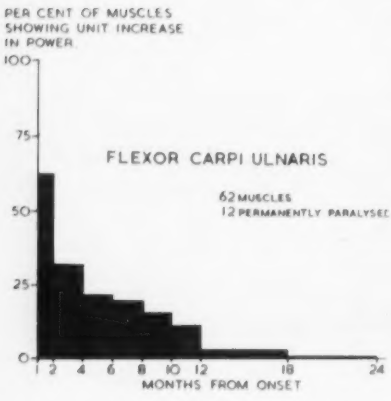

FIG. 6
The rate of recovery in the flexor carpi ulnaris.

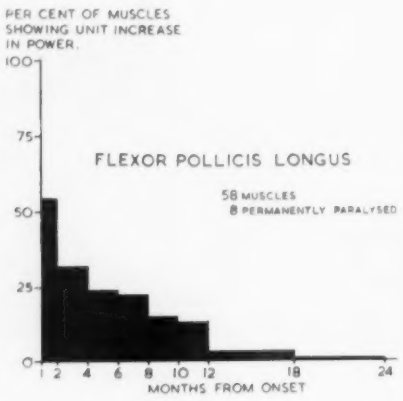

FIG. 7
The rate of recovery in the flexor pollicis longus.

FIG. 8
The rate of recovery in the deltoid muscle.

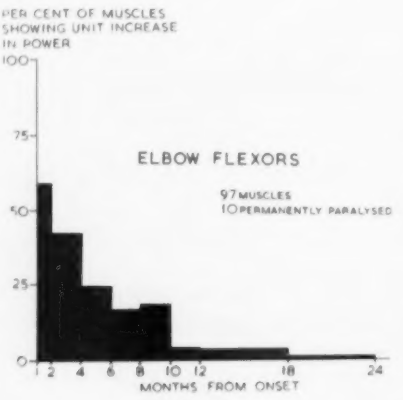

FIG. 9
The rate of recovery in the elbow flexors.

the peronei appeared to recover better than the tibiales, and the gluteus maximus better than the quadriceps.

The reason for this impression can be seen from inspection of Table I: those muscles that show poor clinical recovery include a high proportion of permanently paralysed muscles among their number, whereas those that show apparently better recovery include fewer permanently paralysed muscles.

THE RATE OF RECOVERY IN THE MUSCLES OF THE UPPER LIMB

The same calculations were made to determine the rate of recovery in the 1,905 upper limb muscles. The results, shown in histogram form in Figure 5, are very like those for the lower limb, though the rate of recovery is about 5 per cent per month higher in the upper limb at any time interval up to the tenth month.

The individual muscles of the upper limb show parallel rates of recovery, though with slightly more variation between each other than was present in the lower limb muscles. The results in the forearm and hand muscles, represented by flexor carpi ulnaris (Fig. 6) and flexor pollicis longus (Fig. 7) follow the pattern of the upper limb muscles as a whole (Fig. 5); but in the shoulder musculature, particularly in the deltoid muscle (Fig. 8), the rate of recovery falls suddenly at the fourth month and then increases slightly up to the tenth month, after which it falls to less than 7 per cent per month. The upper arm muscles, represented by the elbow flexors (Fig. 9) show curves of recovery intermediate between the two patterns.

Abduction splints were applied in many cases when paralysis in the upper limb affected the abductors of the shoulder. It is possible that the retarded recovery in the deltoid muscle between the fourth and eighth months was due to the wearing of these splints (Knowleden and Sharrard 1955). The enhanced recovery between the eighth and tenth months coincides with the time at which many of the splints were discarded.

As in the lower limb, the apparently poor power of recovery in such muscles as the thenar group of muscles and the deltoid muscle depends on the larger proportion of permanently paralysed muscles among their number (Table II).

RECOVERY IN RELATION TO AGE

The curve of recovery shown in Figure 1 is an average curve for all patients of all ages. Some patients appeared to recover more rapidly than others, completing the bulk of their recovery by the tenth month. Others recovered more slowly than the average, but continued

TABLE III

DISTRIBUTION OF 142 PATIENTS IN AGE GROUPS

Age group	Number of patients
0–2 years	27
2–4 years	30
4–10 years	27
10–20 years	23
Over 20 years	35

to make significant gains in power up to or beyond the twelfth month. These variants were independent of the extent or distribution of the paralysis, but they did appear to be related, at any rate in part, to age.

A division of patients was made into five age groups, each containing a comparable number of patients (Table III). The rate of recovery was calculated in each group.

The four to ten years group and, to a less extent, the two to four years group show curves

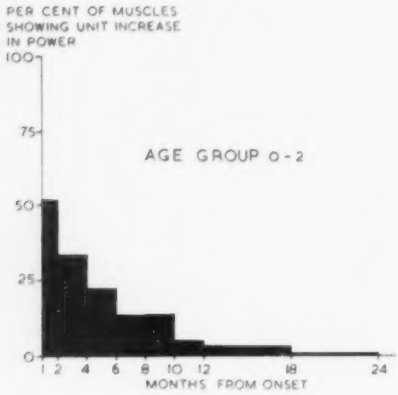

FIG. 10

The rate of recovery in the under two years age group.

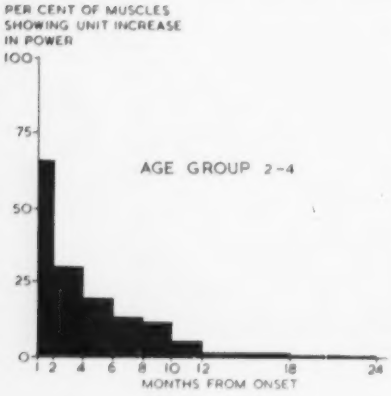

FIG. 11

The rate of recovery in the two to four years age group.

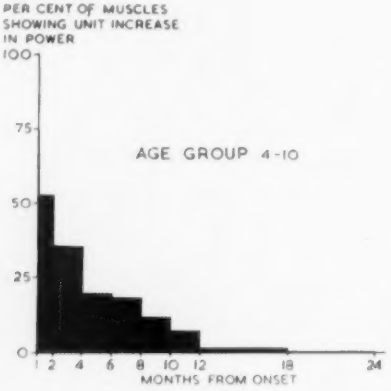

FIG. 12

The rate of recovery in the four to ten years age group.

FIG. 13

The rate of recovery in the ten to twenty years age group.

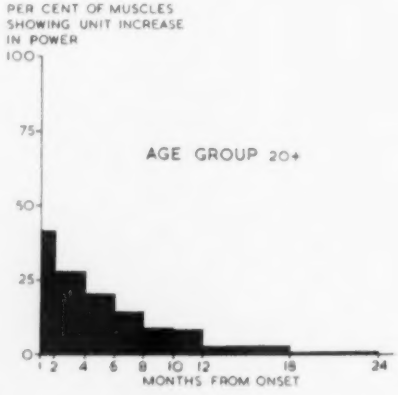

FIG. 14

The rate of recovery in the over twenty years age group.

of rapid recovery (Figs. 11 and 12) whereas the over twenty years group (Fig. 14) shows slow recovery and the ten to twenty years group has an average recovery (Fig. 13).

The curve of recovery in the under two years group (Fig. 10) is especially interesting because infants of this age cannot co-operate in formal muscle re-education. Yet the pattern of recovery does not differ significantly from that in any other group and the total amount of recovery is as great as, if not greater than, that in the others. The resilience and adaptability of the infant and the fact that muscle education is one of his natural tasks probably account for this.

THE PROGNOSIS FOR RECOVERY IN PARTLY PARALYSED MUSCLES

Lower limb—In the calculation of the rate of recovery it has not been necessary to consider the actual grade of a muscle because the unit of recovery has been an increase in grade.

To estimate the amount of recovery in the muscles of the lower limb, all muscles that were assessed at grade 1 at the first (one month) muscle test were reviewed to determine their grade at the end of the investigation. Muscles assessed at grade 2, 3, 4 and 5 at one month were treated in the same way. The results (Fig. 15) show that, although a small proportion of muscles of each grade failed to improve at all, and a few gained by as much as four grades, most muscles improved by one, two or three grades. After making allowance for the fact that muscles that begin at grade 5 cannot increase in power by more than one grade, an average increase of about two grades was made by all muscles from their level of paralysis at the one month muscle test, whether their grade at that time was 1, 2, 3 or 4 (Table IV).

A fresh calculation was then made to discover the improvement in power of the lower limb muscles from the grade at which they had been assessed at the second (two month) muscle test. Again, a small proportion failed to improve or made a spectacular increase in power, but most improved by one or two grades (Fig. 16). An average increase of about one and a half grades was made by muscles of all levels of paralysis except those that commenced at grade 5 (Table V).

After the fourth month about half of the muscles of any given grade improved by one grade, a quarter of them failed to improve, and a quarter improved by two grades (Fig. 17). The average increase was one grade (Table VI). After the sixth month almost half of the muscles improved by one grade but a larger proportion, over a third, failed to improve and a correspondingly smaller proportion improved by two grades (Fig. 18). The average increase was three-quarters of a grade (Table VII).

The uniformity of recovery is such that it is possible to predict the further recovery that may be expected in a muscle from a knowledge of its grade at any time after one month from the onset of the paralysis. On the average the final grade of a muscle in the lower limb may be estimated by adding 2 to its grade at one month, 1·5 to its grade at two months, 1 to its grade at four months, and 0·75 to its grade at six months.

In clinical practice these figures have proved to be useful guides to prognosis. But it must be appreciated that they are mean values and that the amount of recovery can vary within the limits shown in Figures 15 to 18.

Upper limb—The results of the same analysis for muscles of the upper limb are given in Figures 19 to 22 and Tables VIII to XI. As in the lower limb muscles, there is more variation in the final grade reached by muscles from the tests at one month and two months than from those at four months and six months.

But the amounts of recovery are somewhat greater than the corresponding ones for the lower limb muscles. On the average, the final grade of a muscle in the upper limb may be estimated by adding 2·5 to its grade at one month, 2 to its grade at two months, 1·5 to its grade at four months and 1 to its grade at six months.

TABLE IV

AMOUNT OF RECOVERY MADE BY LOWER LIMB MUSCLES FROM ONE MONTH AFTER THE ONSET OF THE PARALYSIS

Grade at one month	Mean grade at twenty-four months	Mean increase in grade
0*	2·2	2·2
1	3·0	2·0
2	4·2	2·2
3	5·2	2·2
4	5·9	1·9
5	6·0	1·0

TABLE V

AMOUNT OF RECOVERY MADE BY LOWER LIMB MUSCLES FROM TWO MONTHS AFTER THE ONSET OF THE PARALYSIS

Grade at two months	Mean grade at twenty-four months	Mean increase in grade
0*	1·8	1·8
1	2·5	1·5
2	3·5	1·5
3	4·7	1·7
4	5·6	1·6
5	6·0	1·0

TABLE VI

AMOUNT OF RECOVERY MADE BY LOWER LIMB MUSCLES FROM FOUR MONTHS AFTER THE ONSET OF THE PARALYSIS

Grade at four months	Mean grade at twenty-four months	Mean increase in grade
0*	1·5	1·5
1	2·0	1·0
2	3·0	1·0
3	4·0	1·0
4	5·2	1·2
5	5·8	0·8

TABLE VII

AMOUNT OF RECOVERY MADE BY LOWER LIMB MUSCLES FROM SIX MONTHS AFTER THE ONSET OF THE PARALYSIS

Grade at six months	Mean grade at twenty-four months	Mean increase in grade
0*	1·3	1·3
1	1·8	0·8
2	2·7	0·7
3	3·7	0·7
4	4·9	0·9
5	5·6	0·6

* Excluding permanently paralysed muscles.

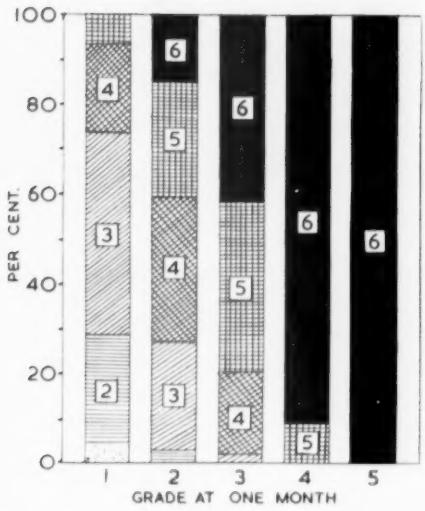

FIG. 15

Lower limb. The final grade of muscles assessed at one month from the onset.

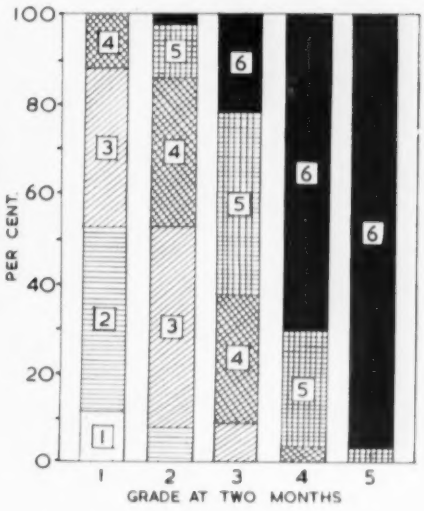

FIG. 16

Lower limb. The final grade of muscles assessed at two months from the onset.

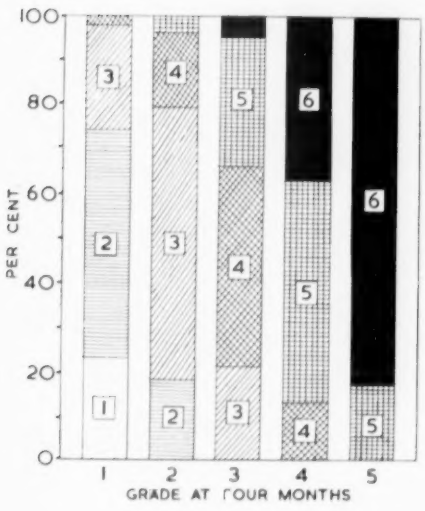

FIG. 17

Lower limb. The final grade of muscles assessed at four months from the onset.

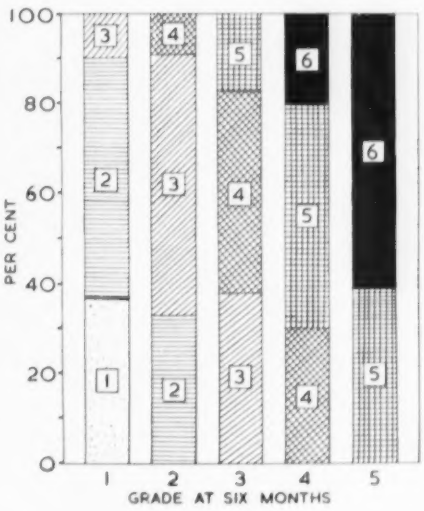

FIG. 18

Lower limb. The final grade of muscles assessed at six months from the onset.

TABLE VIII

AMOUNT OF RECOVERY MADE BY UPPER LIMB MUSCLES FROM ONE MONTH AFTER THE ONSET OF THE PARALYSIS

Grade at one month	Mean grade at twenty-four months	Mean increase in grade
0*	2·97	2·97
1	3·2	2·2
2	4·4	2·4
3	5·37	2·37
4	5·92	1·92
5	6·0	1·0

TABLE IX

AMOUNT OF RECOVERY MADE BY UPPER LIMB MUSCLES FROM TWO MONTHS AFTER THE ONSET OF THE PARALYSIS

Grade at two months	Mean grade at twenty-four months	Mean increase in grade
0*	2·5	2·5
1	3·0	2·0
2	3·9	1·9
3	5·0	2·0
4	5·8	1·8
5	6·0	1·0

TABLE X

AMOUNT OF RECOVERY MADE BY UPPER LIMB MUSCLES FROM FOUR MONTHS AFTER THE ONSET OF THE PARALYSIS

Grade at four months	Mean grade at twenty-four months	Mean increase in grade
0*	2·0	2·0
1	2·2	1·2
2	3·2	1·2
3	4·4	1·4
4	5·4	1·4
5	5·9	0·9

TABLE XI

AMOUNT OF RECOVERY MADE BY UPPER LIMB MUSCLES FROM SIX MONTHS AFTER THE ONSET OF THE PARALYSIS

Grade at six months	Mean grade at twenty-four months	Mean increase in grade
0*	1·7	1·7
1	2·3	1·3
2	2·9	0·9
3	4·0	1·0
4	5·1	1·1
5	5·8	0·8

* Excluding permanently paralysed muscles.

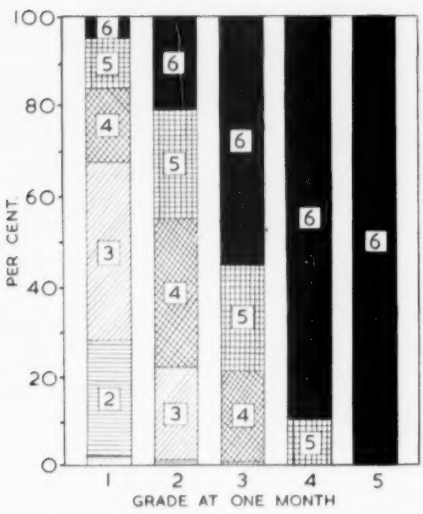

FIG. 19

Upper limb. The final grade of muscles assessed at one month from the onset.

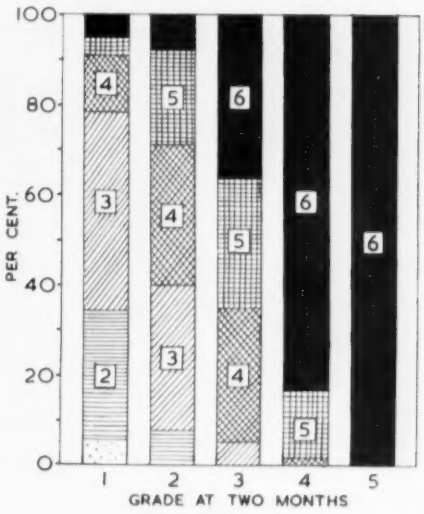

FIG. 20

Upper limb. The final grade of muscles assessed at two months from the onset.

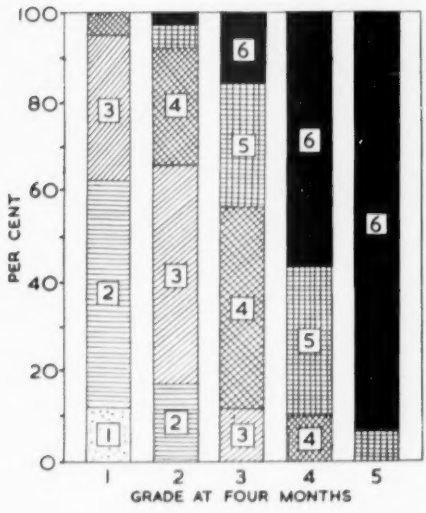

FIG. 21

Upper limb. The final grade of muscles assessed at four months from the onset.

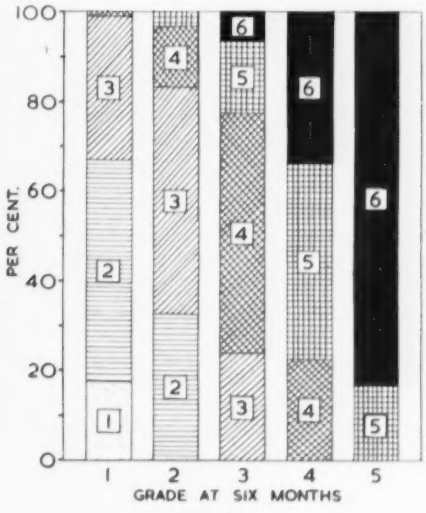

FIG. 22

Upper limb. The final grade of muscles assessed at six months from the onset.

Whether these figures represent a real superiority in the recovery of muscles in the upper limb over those in the lower limb, or whether they arise from differences in the technique of manual muscle testing in the limbs, is a question that this investigation cannot answer.

THE PROGNOSIS FOR RECOVERY IN COMPLETELY PARALYSED MUSCLES

The further recovery of a muscle in which clinical activity can be detected during the first six months after the onset of the paralysis is almost certain. The presence of active muscle fibres implies the existence of intact motor nerve cells in the anterior horn of the spinal cord. A muscle that is clinically completely paralysed may or may not possess a few intact motor nerve cells. If it does, then, given time and favourable circumstances for muscle action and re-education, it will recover. If all the motor nerve cells that supply it have been destroyed it cannot recover.

Figures 23 and 24 show how the passage of time makes the prognosis of completely

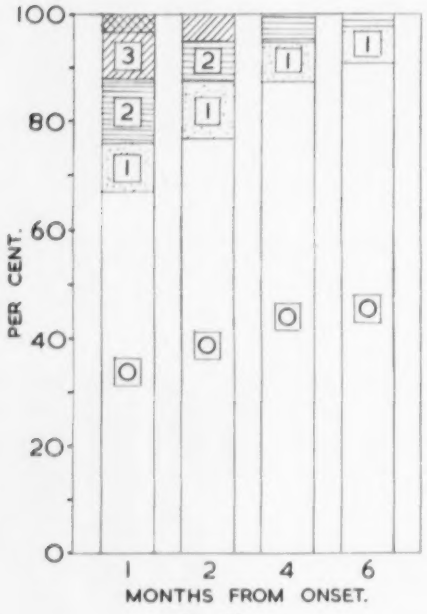

FIG. 23

Lower limb. The final grade of muscles completely paralysed at one, two, four and six months from the onset.

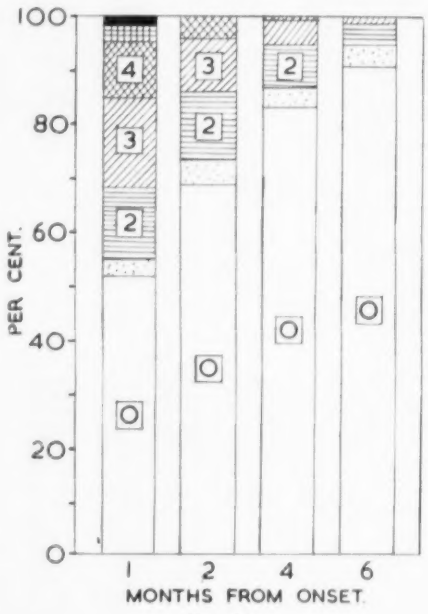

FIG. 24

Upper limb. The final grade of muscles completely paralysed at one, two, four and six months from the onset.

paralysed muscles more certain. One month after the onset a completely paralysed muscle had rather less than an even chance of showing some recovery; but after six months over 90 per cent of the muscles that were still clinically inactive remained completely paralysed, and, of those that did recover, less than half achieved more than a flicker of contraction. If, at any point of time, a completely paralysed muscle did show any return of activity, its prognosis then followed that described for partly paralysed muscles.

Though in many patients it may be appropriate to observe the progress of a completely paralysed muscle for six months to determine its prognosis, it may be important, particularly in a fairly severely paralysed limb, to have some early indication of the likelihood of recovery so that decisions may be made about the management of the paralysis.

Electrical tests, of which Brooks's (1953) percutaneous method of demonstrating nerve

conductivity is probably the most accurate, may show the presence of intact axons supplying muscles that are clinically inactive, but they require appropriate apparatus and expert knowledge, and are not applicable to all muscles.

The destruction of motor cells in the spinal cord has been shown to occur regionally and focally (Elliott 1942, Sharrard 1953). A consequence of this is that the complete loss of all the motor cells supplying a muscle may sometimes be inferred from the presence of complete paralysis in muscles supplied by the same and adjoining spinal cord segments. Thus, the presence of complete paralysis of the quadriceps (L.2, 3, 4) and tibialis posterior (L.4, 5) indicates that a tibialis anterior (L.4) which is also completely paralysed is very unlikely to recover.

A special example of this principle applies to the completely paralysed limb in which the poor prognosis for recovery in any one of its muscles has already been recognised (Seddon 1949). In such cases the anterior horns of the spinal cord are often completely devoid of motor cells. In this series there were twenty-two lower limbs in which no muscle was active one month after the onset (except, in a few cases, for a feeble contraction in the small muscles of the foot). All the muscles in all of these limbs remained completely paralysed at the end of three years. In contrast, in the whole series 33 per cent of muscles that were completely paralysed at one month showed some recovery.

DETERIORATION IN MUSCLE POWER

A loss of power between one muscle test and the next was very uncommon. It was never seen in this series in any of the muscles of the upper limb except in the thenar muscles on three occasions, in each of which power was regained at the subsequent muscle test.

In the lower limb thirty-four instances of deterioration were observed, an incidence of 0·03 per cent. In thirty-one of these the deterioration did not continue; the muscle power either remained stationary at the next muscle test or the original power was regained. The muscles predominantly affected were extensor digitorum longus (five times), extensor hallucis longus (four times), the hip abductors (four times) and tibialis anterior (three times).

Three permanent deteriorations, in which a muscle continued to decrease in power until it became clinically completely paralysed, although initially it had been assessed at grade 2 or grade 3, were observed in tibialis anterior, extensor hallucis longus and extensor digitorum longus. In each of these instances a strong opposing force (muscle pull or gravity) was present.

DISCUSSION

The key to the understanding of muscle recovery in poliomyelitis is the division of the paralysis into a "recoverable" and an "irrecoverable" fraction whose proportions are determined in each case by the site and extent of the motor nerve cell destruction that has occurred during the acute stage of the disease.

It is the irrecoverable fraction that accounts for the variability of the disease upon which Courtney and other writers have commented. This is the factor that governs the general severity of the paralysis in an epidemic, in a patient, or in a limb, and that is responsible for the apparent differences in the power of recovery of different muscles.

When the irrecoverable fraction—that is, the muscles that have suffered complete and permanent paralysis by loss of all the motor nerve cells supplying them—is separated from the remainder the true picture of muscle recovery can be seen. In spite of the relatively coarse and arbitrary method of clinical measurement of muscle power used in this study, the constancy shown in the rates of recovery in muscles of diverse function and size, in patients of all ages, is as remarkable as it is significant.

Whatever the mechanisms of muscle recovery are, whether neural, muscular, or a combination of the two, they must act in the same way and to the same degree in all muscles and in all patients, however mild or severe the paralyses. In this respect the recovery of muscle in poliomyelitis differs from the recovery in peripheral nerve injuries in which factors

such as the quality of the suture line and the distance of the muscle from it determine the outcome. The similarity of the curves of recovery for different muscles and the absence of any sudden spurts of recovery in poliomyelitis suggest that downgrowth of axons from the spinal cord plays no part in recovery.

The fundamental division into recoverable and irrecoverable paralysis defines the correct clinical approach to the treatment and prognosis of poliomyelitis. A patient seen one or two months after the acute disease with a scattered paralysis in which most of the muscles are in grade 2 or grade 3 can confidently be expected to make a fairly complete recovery; indeed if any muscles substantially fail to make the predicted amount of recovery, or show deterioration in power, an inhibiting factor such as an over-strong antagonist or a position of deformity should be suspected.

On the other hand if, after six months of treatment, a limb or a group of muscles in a limb still show complete paralysis, it is unfair to the patient and to the physiotherapist who is treating him to hold out false hopes of recovery. Although a remarkable measure of functional restitution may be achieved by some patients, there is no such thing as a " miracle cure " so far as individual muscles are concerned in a well treated patient.

The results of this investigation may also be applied to aid in planning the treatment of the paralysis in a given limb. For instance, in an adult, a caliper may need to be provided to give stability to the knee (in a child it may be required, in addition, to prevent deformity). In an adult lower limb in which the quadriceps shows a grade of 2 four months after the onset of the paralysis, it would be reasonable to postpone the prescription of a caliper in the expectation of recovery in that muscle to a level (grade 3) sufficient to give stability to the knee joint. In another limb in which the quadriceps is completely paralysed four months after the onset, and muscles supplied by the same spinal segments (the hip adductors and tibialis anterior) are also completely paralysed, treatment should be planned in the expectation that the quadriceps will almost certainly be permanently paralysed.

It is widely believed that if there is an inequality of muscle action at a joint, as for instance when the triceps surae is paralysed to grade 1 and the dorsiflexors of the ankle are acting at grade 3, the process of recovery may restore the balance, the weaker muscles being able to " catch up " with the stronger ones. Unfortunately this never occurs, for both muscle groups recover at the same rate and to the same degree, maintaining the disparity between each other. Even if attempts are made to restore the balance by concentrating treatment on the weaker muscles, any restoration of the balance of power is short-lived. The weaker muscles may in this way be encouraged to reach their greatest power a little sooner than the stronger ones, but, in the course of natural activity after this, the stronger muscles will still continue to recover and the inequality will recur. Appreciation of these facts may be important in deciding whether or not to undertake tendon transplantation.

There is no standard answer to the problem of how long treatment devoted to muscle recovery should be continued. In most cases little further benefit accrues from treatment after nine or ten months; the final residue of gain in muscle power is usually developed in the course of ordinary activity. Occasionally, in a severely paralysed patient, or if the greatest possible effort is required from a muscle in the performance of an important function such as flexion of the elbow, it may be advisable to treat individual muscles for longer than nine months, but never to exceed a total of two years of treatment.

SUMMARY

1. The results of a three-year study of recovery in 3,033 lower limb muscles and 1,905 upper limb muscles in 142 patients are presented.

2. The rate of recovery of partly paralysed muscles is the same in all muscles and muscle groups in the lower or upper limb. Clinical differences in the ability of individual muscles to recover depend upon the proportions of their number that remain permanently paralysed.

3. The rate of recovery is slowest in adults and most rapid in young children.
4. The amount of further recovery to be expected in a muscle can be predicted from a knowledge of its grade at any time after one month from the onset of the paralysis. Fourteen-fifteenths of the total amount of recovery takes place by the beginning of the twelfth month; with rare exceptions individual muscle recovery is complete after twenty-four months.
5. Ninety per cent of muscles that are still completely paralysed after six months remain permanently paralysed.
6. The prognosis of a completely paralysed muscle is related to the level of paralysis in muscles supplied by the same spinal segments.
7. Deterioration in power in a muscle is uncommon and, when it occurs, is associated with the presence of the strong opposing force of antagonist muscles or of gravity.
8. The application of these findings to the management of cases of paralytic acute anterior poliomyelitis is discussed.

I wish to acknowledge with thanks the help and guidance given to me by Mr H. J. Seddon, Director of Studies at the Institute of Orthopaedics, University of London, at which this work was undertaken under a grant from the Medical Research Council. The detailed figures, from which the results given here have been extracted, have been included in a thesis submitted to the University of Sheffield for the degree of Doctor of Medicine (1954).

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THE NATURAL HISTORY OF PLEURAL EFFUSION AND ORTHOPAEDIC TUBERCULOSIS

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Seven years ago two patients with pleural effusion were seen while under treatment for their orthopaedic lesions. They had no evidence of pulmonary disease and their bone focus proved that they had already passed through the stage of haematogenous dissemination. It seemed therefore that something out of the usual sequence of events might be happening, and it was decided to keep a record of all cases observed with pleural effusion, or seen in hospital with a past history of pleural effusion, so that an assessment might be made of the place of this tuberculous complication in the natural history of patients in an orthopaedic hospital.

Since then the records of sixty-three such effusions have been collected. Occasionally more than one effusion or more than one type of effusion has occurred in the same patient.

Table I shows the main sub-divisions of the material.

TABLE I

CIRCUMSTANCES OF EFFUSION

Effusions occurring before the onset of the orthopaedic lesion and without post-primary adult-type pulmonary disease	24
Effusions occurring before the onset of the orthopaedic lesion but probably secondary to post-primary adult-type pulmonary disease	3
Effusions occurring after the onset of the orthopaedic lesion and directly secondary to operative intervention within the thorax	7
Effusions occurring after the onset of the orthopaedic lesion without operative intervention or pulmonary disease	29
	63

The first two groups of twenty-seven patients who had pleural effusion before the onset of symptoms of the orthopaedic lesion provide a useful comparison with the large fourth group. Their details show that, as might be expected, the vast majority of them—twenty-four—were probably the immediate post-primary effusions. The orthopaedic lesions developing subsequently as a result of haematogenous dissemination were again, as might be expected, scattered widely throughout the body.

Table II gives details of the twenty-four cases whose effusions have been presumed due to the post-primary haematogenous dissemination.

Table III shows the details of the three patients with pleural effusions secondary to adult-type pulmonary disease.

The bone and joint lesions developing after post-primary effusions and effusions secondary to adult-type or infiltrative pulmonary disease are summarised in Table IV.

Table V gives the essential histories of the post-operative effusions. They all occurred within one to four weeks of the operation, usually costotransversectomy or antero-lateral decompression of the thoracic spinal cord. In one patient it will be noted that there was a presumptive haematogenous dissemination after a grafting operation on the hip; bilateral pleural effusion occurred three to four weeks later.

TABLE II

DETAILS OF TWENTY-FOUR CASES OF EFFUSION FROM POST-PRIMARY HAEMATOGENOUS DISSEMINATION

Case number	Year of birth	Year of effusion and age at the time	Onset of orthopaedic symptoms	Site of orthopaedic lesion
1	1906	1931—25 years. Right effusion. X-ray 1951 showed right obliterated costo-phrenic angle but normal lung fields	1934	Elbow
2	1916	1945—29 years. Left effusion	December 1948	Hip and lumbar spine. Thoracic spine 9 to 10 lesion discovered June 1949 by cold abscess seen through heart in chest x-ray
3	1916	June 1937—21 years. Left effusion. Had right pulmonary lesion	December 1937	Thoracic spine 9 to 12
4	1919	September 1945—26 years. Right effusion	January 1946	Thoracic spine 7 to 11 with abscess
5	1920	1946—26 years. Left effusion	1948	Lumbar spine 2 and 3
6	1921	1948—27 years	1950	Lumbar spine 4 and 5
7	1921	1944—23 years. Left effusion	1952	Ankle
8	1921	April 1946—25 years. Left effusion	December 1947	Lumbo-sacral spine and sacro-iliac joint
9	1922	1946—24 years. Left effusion plus erythema nodosum. 1949—27 years. Right effusion (see Table VI) and x-ray + bone disease	1948	Thoracic spine 8 and 9
10	1922	1947—25 years. Left effusion. 1948—26 years. Right effusion. Infiltration, bilateral apical, in x-rays 1950	1948	Sternum. Hip
11	1923	1946—23 years. Right effusion. 1947—Apical cavity. 1948—Miliary	1950	Knee
12	1924	1939—15 years	1948	Lumbar spine 4 and 5
13	1926	1941—15 years. Chest x-ray 1951 showed old right lung primary	1951	Lumbar spine 4 and 5
14	1926	Obliterated right costo-phrenic angle found nine months after onset of knee. No history. Presumed old	1942	Knee
15	1927	April 1951—24 years	March 1952	Lumbar spine 2 and 3
16	1927	February 1950—23 years	April 1952	Lumbar spine
17	1929	July 1932—3 years. Right effusion	July 1933	Discovered thoracic spine 6 and 7 with abscess and lumbar spine 4 lesion
18	1931	1947—16 years. Right and left pleural effusions and ascites	1951	Lumbar spine 5 and sacral spine 1
19	1931	1944—13 years	1948	Hip
20	1934	November 1949—15 years. Right effusion. June 1950—Pericarditis	December 1950	Thoracic spine 12 and lumbar spine 1

TABLE II—*continued*

DETAILS OF TWENTY-FOUR CASES OF EFFUSION FROM POST-PRIMARY HAEMATOGENOUS DISSEMINATION

Case number	Year of birth	Year of effusion and age at the time	Onset of orthopaedic symptoms	Site of orthopaedic lesion
21	June 1948	Small right effusion with primary on same side	November 1949	Shoulder
22	1949	1951—2 years. Right effusion plus soft right primary	1951	Right subtalar joint
23	1949	June 1951—2 years. Right inter-lobar effusion. Later right calcified primary seen	August 1951	Thoraco-lumbar spine
24	—	Effusion before symptoms. Details unobtainable	—	Lumbar spine 4 and 5

TABLE III

DETAILS OF THREE PATIENTS WITH EFFUSION SECONDARY TO ADULT-TYPE PULMONARY DISEASE

Case number	Year of birth	Clinical details
25	1881	Phthisis 1915 and tuberculous greater trochanter. 1953: Spread from greater trochanter to hip joint. Chest x-ray: Old left pleurisy and calcified plaques and bilateral apical old disease
26	1913	December 1947: Bilateral infiltration and left effusion. Onset of orthopaedic symptoms: September 1948. Site of orthopaedic lesion. Right sacro-iliac joint. Thoracic spine normal
27	1945	Tuberculous hip and shoulder 1945, and massive primary in right. Right pleural effusion 1947. Tuberculous empyema drained 1949. No bone source

TABLE IV

SITES OF THIRTY-THREE LESIONS IN TWENTY-SEVEN PATIENTS WITH SKELETAL DISEASE OCCURRING AFTER POST-PRIMARY EFFUSIONS OR EFFUSIONS SECONDARY TO ADULT-TYPE PULMONARY DISEASE

Spine:	
Thoracic	5
Thoraco-lumbar	2
Lumbar	9
Lumbo-sacral	2
Hip	4
Ankle	1
Elbow	1
Sacro-iliac joint	2
Sternum	1
Knee	2
Shoulder	2
Subtalar joint	1
Greater trochanter	1
	33

TABLE V

DETAILS OF SEVEN CASES OF POST-OPERATIVE PLEURAL EFFUSION

Case number	Year of birth	Clinical details
28	1905	Right effusion ten days after right costotransversectomy 1953 (lung previously clear)
29	1908	Left costotransversectomy January 1952; three weeks later slightly bloodstained lymphocytic sterile left effusion. March 1952 right antero-lateral decompression; five days later a right sterile lymphocytic effusion.
30	1921	Left effusion two weeks after left costotransversectomy 1948.
31	1935	Left effusion one week after left costotransversectomy 1948
32	1937	Effusion on same side few days after antero-lateral decompression 1952. Pleura was punctured
33	1920	Tuberculous hip 1941. Grafted 1943 Bilateral lymphocytic sterile effusions three to four weeks after operation. No evidence of spinal disease clinically or in chest x-rays. Presumed haematogenous dissemination.
34	1909	Left effusion in artificial pneumothorax 1938. Tuberculous spine 1942

PLEURAL EFFUSIONS OCCURRING AFTER THE ORTHOPAEDIC LESION AND WITHOUT PULMONARY DISEASE OR LOCAL OPERATIVE INTERVENTION

These twenty-nine patients will be seen in Table VI in three groups. The first and largest consists of twenty-three cases. All of these developed their pleural effusion either after the onset of symptoms from a thoracic or thoraco-lumbar tuberculous spondylitis with cold abscess, or actually during the time spent in hospital for treatment of this condition. The

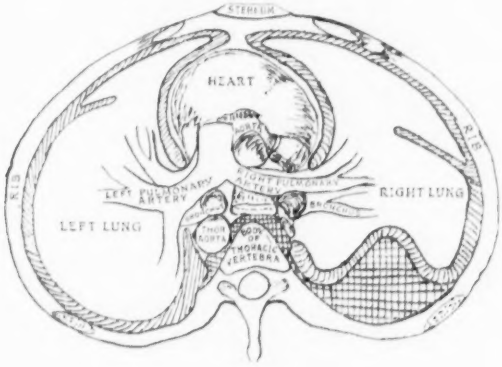

FIG. 1

To show the relationship between the paravertebral region and the outside of the parietal pleura. On the right side an extra-pleural lateral extension of an abscess is shown pushing the pleura and lung forwards as described in the text. The pleural space is shown shaded and the paravertebral abscess cross-hatched.

absence of pulmonary disease makes the fact that their orthopaedic lesion in every case involved the thoracic spine, and therefore lay immediately outside the parietal pleura, highly significant (Fig. 1).

Two more patients, the second group in Table VI, also had thoracic spinal disease but

TABLE VI

DETAILS OF TWENTY-NINE CASES IN WHICH PLEURAL EFFUSION OCCURRED AFTER THE ORTHOPAEDIC LESION

Case number	Year of birth	Year of effusion and age at the time	Onset of orthopaedic symptoms	Site of orthopaedic lesion
35	1892	January 1952—60 years. Transient small effusion	January 1951	Thoracic spine 11 and 12 proved December 1951 + abscess
36	1903	Nursed tuberculous sister 1924 to 1930 (died). Pleural effusion left 1933 (30 years) + pain lower ribs girdling both sides. 1936 discovered thoracic spinal disease with cold abscess mainly projecting to left		
37	1905	42 years. Left pleural effusion four months after costotransversectomy 1947. (Too late for post-operative causation)		
38	1906	December 1937—31 years. Right effusion	January 1937, after fall	Thoracic spine 8 to 12 + abscess
39	1913	August 1952—39 years	May 1952	Thoracic spine 10 to 12 + abscess. + Tarsus
40	1916	November 1946—30 years. Right effusion proved tuberculous. Left dry pleurisy	1943 1946	Thoracic spine 6 and 7 + abscess Lumbar spine 2 to 5
41	1917	Tuberculous thoracic spine 7 to 12 + abscess early 1952. Right costotransversectomy January 1953. Right effusion July 1953 (36 years), guinea pig +, and left August 1953 (too late for post-operative causation).		
42	1917	December 1942—25 years	Back symptoms March 1943; x-rays December 1943 show old thoracic spine 12 and lumbar spine 1 tuberculous disease and bony bridging	
43	1918	February 1946—28 years. When in hospital for treatment of spine	1941	Thoracic spine 7 to 11
44	1919	July 1949—30 years. Left effusion	March 1947	Thoracic spine 8 and 9 x-ray + March 1948
45	1919	September 1945—26 years. Right effusion (post-primary) and recurrence right July 1947 and left May 1948, both tuberculous +	January 1946	Thoracic spine 7 to 11 + abscess
46	1920	28 years. Left effusion approximately 1948 in Stanmore with mid-thoracic disease		
47	1922	July 1950—28 years. Right effusion. Lungs normal. Abscess visible	Under treatment March 1950 to December 1952	Thoracic spine 12 and lumbar spine 1
48	1922	24 years. Erythema nodosum and left effusion 1946 (post-primary). Right effusion 1949 (secondary to abscess)	1948	Thoracic spine 8 and 9 + abscess
49	1935	September 1947—12 years. Lymphocytic right effusion	July 1947	Thoracic spine 9 to 11 + abscess
50	1941	5 years. May 1946 left pleural effusion and at same time found to have large kyphos and abscess mid-thoracic spine		
51	1947	April 1952—5 years	—	Thoracic spine 9. Tuberculosis + abscess found June 1952 and must have been present in April 1952
52	—	Costotransversectomy October 1945. Pleural effusion March 1946	December 1943	Thoracic spine 8 and 9 + abscess
53	1925	1944—19 years. Pleural effusion left and Pott's disease in Army	—	Thoracic spine 7 to 12
54	1920	July 1945—25 years. Right effusion	March 1945	Thoracic spine 10 to 12
55	1924	October 1953—29 years	August 1946	Thoracic spine 8 to 11

TABLE VI *continued*

DETAILS OF TWENTY-NINE CASES IN WHICH PLEURAL EFFUSION OCCURRED AFTER THE ORTHOPAEDIC LESION

Case number	Year of birth	Year of effusion and age at the time	Onset of orthopaedic symptoms	Site of orthopaedic lesion
56	1935	September 1947—12 years. While in hospital for spine	July 1947	Thoracic spine
57	1897	February 1947—50 years. Right. July 1947. Left	July 1947 weakness of the legs and pins and needles from the waist downwards. July 1948 readmitted for increasing paraplegia and discovered to have tuberculous thoracic spine 11 and 12 with cold abscess. Must have been present July 1947 at least.	
58	1905	February 1950—45 years. Right	February 1951	Discovered radiologically to have thoracic 9 and 10 lesion + abscess. No symptoms referable to lesion and ? old. Age suggests effusion *not* post-primary
59	1920	31 years. 1949—Soft lesion above right costo-phrenic angle. 1950—Soft lesions right apex and right lower zone. Cold abscess visible through heart in chest film. January 1951—Pain in the back began. September 1951—Right pleural effusion. It is not certain whether in this case the pleural effusion was secondary to the pulmonary lesion or the cold abscess, though the fact that the abscess had some months previously tracked round to the right and pointed between the ribs strongly suggests the latter		
60	1929	May 1948—19 years	March 1947	Sacro-iliac joint. Thoracic spine never x-rayed
61	1881	1915—Gland in neck. 1931—Sacro-iliac joint lesion. August 1948—Lumbar spine 2 to 4, Addison's disease and tuberculous sacro-iliac joint. December 1948—67 years. Clear left effusion. November 1949—Right effusion. Meningitis, ? ascending, ? haematogenous; died February 1950. No post-mortem		
62	1921	Admitted April 1944: lumbar spine 4 and 5. Left pleural effusion September 1944—23 years. No evidence of pulmonary or thoracic spinal disease		
63	1926	March 1947—21 years. Left effusion while in hospital for tuberculous lumbar spine 3 and 4. No evidence of pulmonary or thoracic disease.		

there was some uncertainty as to the origin of the effusion and though it may have been due to the paravertebral abscess the claim could be made with less certainty.

Case 58—This patient suffered a right-sided effusion at the age of forty-five and was accidentally discovered to have a vertebral lesion at T.9–10 with abscess a year later. There were no symptoms referable to this lesion and it may well have been old and present at the time of her effusion. Her age itself suggests that the effusion was not immediately post-primary.

Case 59—This patient has also been listed in this separate group because she did have pulmonary lesions in the right chest from February 1949 onwards and developed a right pleural effusion in September 1951. The cold abscess could be seen, however, through the heart shadow in the chest radiograph as far back as May 1950 and could well have been the cause of her effusion.

The third group of four patients who developed a pleural effusion after their orthopaedic lesion without evidence either of pulmonary lesions or of thoracic spinal disease contains one patient (Case 60) whose thoracic spine was never in fact radiographed, one (Case 61) who had bilateral effusions fifteen months (left) and three months (right) before death and actually died of meningitis. This may have been ascending or haematogenous. There was no post-mortem examination. The last two cases (62 and 63) definitely had pleural effusions while in hospital under treatment for lower lumbar spinal disease only, and in each the thoracic spine was radiologically normal.

Of the twenty-nine patients in this group, therefore, twenty-three showed no other apparent reason for effusion than the thoracic spondylitis; in two more very probably the same reason held; in a further two, essential details are lacking and only in a final two did effusion certainly occur during conservative treatment for lumbar spinal disease without evidence of thoracic or pulmonary lesions.

The conclusion that may be drawn from these figures is quite clear: when a pleural effusion occurs before the onset of orthopaedic disease or is clearly secondary to pulmonary

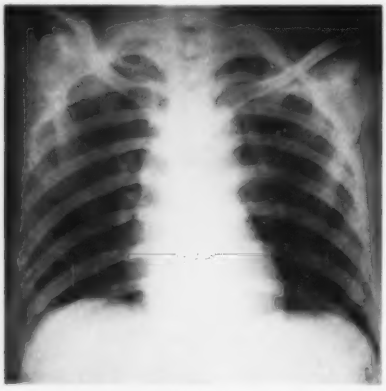

FIG. 2
Case 54—Before effusion.

FIG. 3
Case 54—Right pleural effusion.

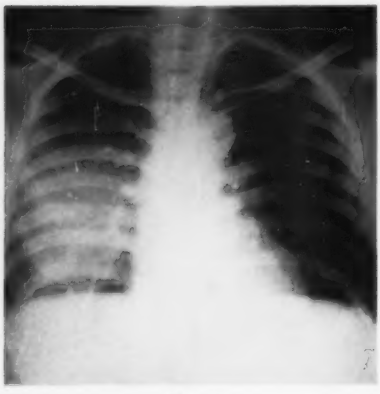

FIG. 4

FIG. 5

Case 54—Lateral extension of cold abscess, seen bulging forwards in the lateral view.

disease then the orthopaedic lesion may be anywhere throughout the skeleton (Table IV). But when the effusion arises after the onset of the orthopaedic disease and without a pulmonary cause or immediately preceding operative intervention in the thorax the lesion will almost certainly involve the thoracic spine. It is difficult to avoid the conclusion that this group of pleural effusions occurring after the period of haematogenous dissemination and without pulmonary disease is, in fact, due to the direct spread through the parietal pleura of tuberculous infection from the paravertebral abscess (Fig. 1).

It was surprising to find that there was little variation in the clinical picture produced by these pleural effusions whatever the cause, except in the amount of effusion and the length of time that it took to absorb. This, of course, might be affected, even in large effusions, by

the decision whether or not to aspirate. In the cases observed, whether they were post-primary, post-operative or during conservative treatment for the orthopaedic lesion, the pyrexia commonly rose and after a plateau gradually fell over a period of three to four weeks. None of the effusions became purulent; the cytology was predominantly lymphocytic. As is common with tuberculous pleural effusions acid-fast bacilli were sometimes grown and sometimes not.

During the time required to collect twenty-five cases of this transpleural infection 145 patients with thoracic or thoraco-lumbar spinal tuberculosis have been seen at this hospital; so in a proportion of approximately one in six a pleural effusion occurred that was not immediately post-primary. Ornstein and Ulmar (1935, 1939) believed a thoracic paravertebral abscess to be a not uncommon cause of unilateral and bilateral pleural effusions. Of the standard text-books upon chest diseases, Marshall and Perry (1952) and Pagel, Simmonds and Macdonald (1953) mention the possibility, whereas other well known works do not, and it has not seemed to the writer from conversations with colleagues that the connection was well recognised. In particular, however, this report is offered not to illustrate the fact that the sequence is possible but that in so high a proportion of effusions occurring after the onset of the bone symptoms this relationship is to be expected. Seddon (1936) recorded a case of rupture of a paravertebral abscess into the pleura giving rise to a large tuberculous empyema, but this is an entirely different and much more rare occurrence.

PLEURAL EFFUSION SECONDARY TO LARGE LATERAL EXTRA-PLEURAL EXTENSION OF PARAVERTEBRAL ABSCESS

There is one sub-group of thoracic paravertebral abscesses which does not appear to have been stressed before. Their behaviour is illustrated in the right side of Figure 1 and the histories in Cases 54, 55 and 56 are given in illustration.

Case 54—This patient developed tuberculosis of the spine at T.10–T.12 in March 1945. Chest radiographs a month later showed normal and equal lung fields with an abscess shadow just visible inside the right border of the heart (Fig. 2). Three months later she developed a right pleural effusion (Fig. 3). Further chest radiographs in July 1948 showed a diffuse shadow occupying the right lower zone, with some translucency both on the medial and lateral sides of it (Fig. 4). This type of shadow, which might well represent an inter-lobar effusion, had been seen before, and lateral radiographs proved that it was in fact due to an abscess bulging forwards from the posterior chest wall (Fig. 5). The abscess was needled three inches from the mid-line in the right lower chest; inspissated pus was withdrawn and guinea-pig inoculation confirmed its tuberculous nature.

Case 55—This patient had tuberculous disease of the spine in 1943 which became temporarily quiescent. Chest radiographs in October 1949 showed normal lung fields and the old abscess shadow could just be seen through the heart shadow (Fig. 6). About August 1953 he developed a total pleural effusion on the left side. Radiographs three months later again showed the shadow with increased translucency both medial and lateral to it (Fig. 7). The lateral radiograph showed a large abscess projecting forwards at the level of the spinal lesion (Fig. 8). Needling about three inches from the mid-line yielded pus, proved tuberculous by guinea-pig inoculation. Repeated aspiration has caused a reduction in size of the abscess. Now, four months after admission, the condition is that shown in Figures 9 and 10.

Case 56—The patient in this case developed tuberculous disease of the spine at T.9–T.11 in July 1947. Two months later he had a right lymphocytic pleural effusion. The chest radiographs taken a month before the effusion developed were clear, except for the central paravertebral abscess which could just be seen through the heart (Fig. 11). Radiographs taken one month after the effusion (Fig. 12) showed a condition like that seen in Case 55. The lateral film again showed a large abscess projecting forwards (Fig. 13). Successive aspirations of large quantities of pus finally reduced the abscess to a very small inspissated collection close to the posterior thoracic wall (Fig. 14).

LYMPHATIC SPREAD FROM PLEURA TO LYMPH NODES AND THENCE TO BONE

A study of pleural effusion and skeletal tuberculosis would be incomplete without discussing the belief that tuberculous pleurisy may be a cause of bone disease by lymphatic spread to ribs and spine. Souligoux (1894) demonstrated the lymphatic connections between the pleura and parasternal lymph nodes, and showed that tuberculous pleurisy, and particularly

FIG. 6
Case 55—Before effusion.

FIG. 7

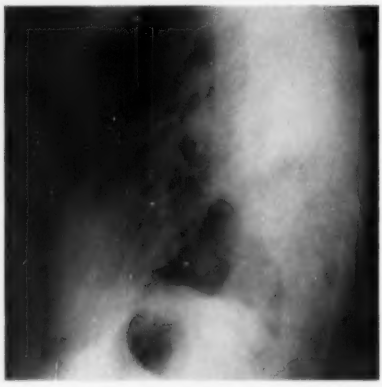

FIG. 8

Case 55—Lateral extension of cold abscess after effusion. The abscess is seen bulging forwards in the lateral view.

FIG. 9

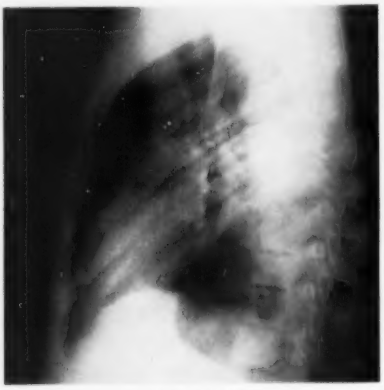

FIG. 10

Case 55—Abscess reduced after repeated aspiration.

empyema, could produce caseous parasternal lymph nodes. Kaufmann (1930, 1931, 1933) followed by showing that cold abscess of the chest wall could arise from these caseous parasternal lymph nodes. But he also suggested that paravertebral cold abscess and disease of the spine could be secondary to caseous disease of the para-aortic lymph glands, especially in the neighbourhood of the crura of the diaphragm. Both Fraser (1913, 1929) and Kaufmann (1936) did demonstrate in a few cases that a caseous tuberculous gland lying against a vertebral

FIG. 11

FIG. 12

Case 56. Figure 11—Before effusion. Figure 12—Lateral extension of abscess after effusion.

FIG. 13

FIG. 14

Case 56. Figure 13—Same as Figure 12, lateral view. Figure 14—Abscess markedly reduced after repeated aspiration.

body could produce superficial caries of the bone by lymphatic spread through the paravertebral ligaments.

Burke (1940, 1940, 1947, 1950) correlated this work and tried to prove more conclusively the connection between pleurisy and tuberculous disease of the ribs and chest wall and of the spine. He was able to show experimentally in guinea pigs that particulate matter—a mixture of colloidal thorium dioxide and finely divided lamp black—or tubercle bacilli in suspension in isotonic saline, when injected into the pleural spaces, would in part at least be transported to the parasternal and the para-aortic lymph nodes. Observations in man, at necropsy, also showed that tubercle bacilli and particulate matter such as siliceous material were conveyed from the pleural spaces to these same two groups of glands.

He suggested therefore that the occurrence of apparently spontaneous cold abscess of the chest wall really followed caseation of lymph nodes, rupture, and direct involvement of the chest wall with eventual burrowing of the cold abscess to the surface. He was not, however, able to demonstrate the production of a cold abscess of either the chest wall or spine in animals secondarily to post-pleuritic infection of lymph nodes; he was only able in 1940 to demonstrate a possible mode of spread of particulate matter.

He had carried the matter somewhat further by 1950 however. Among fourteen patients with abscess of the chest wall six had had previous pleurisy and ten had co-existing pleurisy; but nine of these fourteen patients had pulmonary tuberculosis, which might well have caused the pleurisy directly and he did not state how long before the appearance of the cold abscess the antecedent pleurisy had occurred. Therefore, although the sequence tuberculous pleurisy—caseous parasternal lymph gland—rib or chest wall abscess is not impossible, it cannot be said that Burke has shown that it occurs frequently or in what proportion it does occur. Burke also cited forty-five cases of Pott's disease studied from the Royal Victoria Hospital and the Montreal Neurological Institution between 1943 and 1950 in an attempt to prove that disease of the paravertebral space and the spine readily occurs as a result of caseation in overlying para-aortic glands. He listed eighteen patients with antecedent and twenty-one with co-existent pleurisy, some with both. But thirty of these also had pulmonary tuberculosis, which again bedevils the argument. Of the only four patients who had a co-existent pleurisy and Pott's disease, without pulmonary tuberculosis, three had disease in the mid-thoracic region and one had involvement of the first and second lumbar vertebrae with a paravertebral abscess which might well have spread both above and below the actual bone lesion. All these four cases would therefore confirm the findings in this paper: that co-existent effusion and Pott's disease affects one particular part of the spine—namely that in which the paravertebral abscess is in contact with the parietal pleura—and that the spread is from spine to pleura and not from pleura to spine. Furthermore, if the infection commonly spread from pleura to glands and thus to spine, the back spread to pleura again with consequent effusion would be most unlikely instead of frequent, because previous pleurisy would probably have produced adhesion between the parietal and visceral layers.

It cannot be denied from Burke's own work, and that of the other writers quoted, that lymphatic spread from an overlying caseous gland through the paravertebral ligaments may occasionally produce superficial disease of bone. But Burke goes on to say: " facts clearly point to the conclusion that Pott's disease is in many instances lymph-borne from tuberculous pleuritis "; and also claims that the experimental, anatomical and clinical findings recorded seem clearly to point to the conclusion that most cases of Pott's disease are the result of lymph-borne rather than blood-borne dissemination of tubercle bacilli. These statements are simply not justified by figures. The large number of cases of tuberculosis of bone in the limbs must be produced by haematogenous dissemination. It is equally likely that the same etiology is common in the axial skeleton and that Burke's conclusion upon Pott's disease is, in our present state of knowledge, an example of special pleading.

SUMMARY

1. Details are given of sixty-three consecutive cases with a history of pleural effusion seen at an orthopaedic hospital.

2. Twenty-four of these were post-primary effusions occurring before the onset of symptoms of the orthopaedic lesion. The bone and joint lesions ultimately developing in this group of patients were widely scattered throughout the skeleton.

3. Three others were secondary to adult-type pulmonary lesions.

4. Thirty-six patients had a pleural effusion after the beginning of their orthopaedic tuberculous history. Seven were certainly secondary to operative intervention, six in the

thorax near the parietal pleura (costotransversectomy or antero-lateral decompression of the cord) and one from a haematogenous dissemination after fusion of a hip joint.

5. The remainder of this group with pleural effusion during the history of their orthopaedic tuberculous disease numbered twenty-nine. Of these, twenty-five suffered from disease of the thoracic spine; in two more details are defective. Only two definitely had neither pulmonary nor thoracic spinal disease; their lesions were in the lumbar spine.

The conclusion is drawn that the overwhelmingly common cause of pleural effusion in patients with orthopaedic tuberculosis who have normal lungs and have not recently suffered spinal decompression is transpleural infection from thoracic spinal disease and that the sequence is by no means rare. It had occurred in approximately one in six of 145 patients with thoracic Pott's disease seen during this investigation.

6. Details are given of a group of cases with thoracic paravertebral abscess tracking laterally. When the abscess is well clear of the spine and spinal ligaments it may project forwards and radiologically it may appear in the antero-posterior chest film as a shadow in the middle of one or other lung field rather than as a shadow obviously connected with the central paravertebral abscess. Aspiration will yield pus from this posterior extra-pleural abscess extension.

7. The belief that Pott's disease most commonly follows direct spread from caseous para-aortic glands secondary to tuberculous pleurisy is discussed. It is concluded that the evidence is insufficient for so sweeping a statement.

The patients in this survey have been under the care of my surgical colleagues on the staff of the Royal National Orthopaedic Hospital and I am grateful to them, as always, for their co-operation; and also to Dr E. H. Allen, Radiologist, and Dr. C. H. Lack, Bacteriologist to the hospital.

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SCOLIOSIS: OPERATIVE CORRECTION OF FIXED CURVES

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This paper is a preliminary report on an attempt to obtain a quicker and more effective correction of spinal curvature in scoliosis before fusion than the Risser jacket (Hibbs, Risser and Ferguson 1931) and similar methods of gradual correction in plaster can afford. A claim is also made that some correction is also obtained in cases that at first sight appear to be uncorrectible. The method also prevents any serious relapse pending consolidation after correction and a fusion operation.

Correction of the lateral curve in a Risser jacket is an indirect procedure. As the trunk is flexed laterally the spine bends with it and tends to maintain its more or less central position; correction is excellent if the curve is not too great and reasonably mobile. In severe and fixed curves, however, the effect of lateral wedging is to force the ribs in without materially altering the spinal curvature, so that in the end the spine may be close up against the lateral thoracic wall, with little or no correction of the curve. Again, a review of cases treated by bone grafting alone suggests that relapse may occur slowly over a long period even though the consolidation seen in radiographs may appear adequate.

PRINCIPLES OF THE METHOD

It is in the hope of overcoming these difficulties that the method to be described was devised. The curve is forcibly corrected by lengthening its concavity by means of an expanding device made of an inert metal, which can be left in position afterwards for an indefinite period and so allow a satisfactory fusion operation to be performed and give plenty of time for good consolidation. The device or jack takes the form of two Y-shaped pieces, the stems threaded left and right handed and the limbs tapering and pointed at the extremities (Fig. 1). They

Fig. 1

are angled about 30 degrees at the junction of stem and limbs, and connected together by a threaded sleeve which can be turned by a small tommy bar. At operation the notch between the limbs engages the base of a transverse process at its junction with the vertebral body, and the angulation allows the extremities to point towards the body and drive in as the Y-pieces are separated. Before operation a complete radiological examination of the spine is required; the siting of the primary and secondary curves must be determined, and an estimate of the correction theoretically possible in the primary curve made, after measurement of the angles of the secondary curves on bending to right and to left. Any improvement on traction should also be noted. The placing of the jack is decided; it is usually put between the transverse processes of the vertebrae at the top and bottom of the major curve. If, however, it is obvious from a study of the shape of the transverse processes that a much firmer hold is available at a level higher or lower, then this should be chosen.

OPERATIVE TECHNIQUE

The operation is performed in much the same way as a spinal fusion after correction in a Risser jacket. The spinous processes and laminae and pedicles are exposed on the concave side of the curve, and, in the centre of the curve, parts of the ribs will be exposed as well. On the convex side processes and laminae only are exposed. The junction of the transverse process and body at the chosen levels is clearly defined. This may not be easy. The distortion of the vertebrae may be extreme, especially in cases of congenital origin, and the processes may slope away from the body at awkward angles. The vertebral body may have to be

FIG. 2

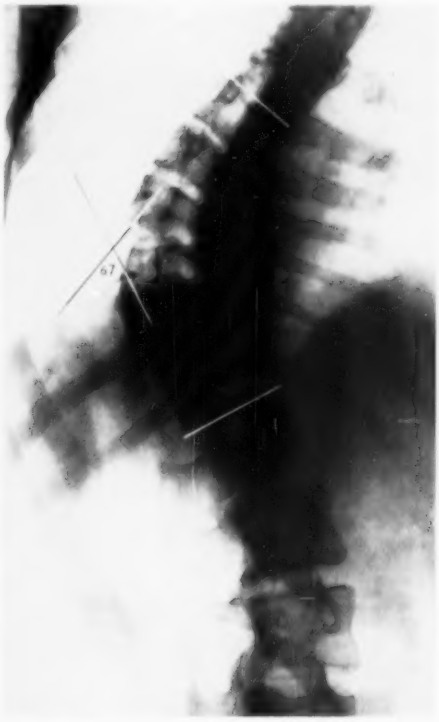

FIG. 3

Case 9—Idiopathic scoliosis. Age eighteen. Figure 2—Before correction. Figure 3—After correction.

notched with a small gouge to obtain an adequate anchorage for the jack, especially if the transverse processes are slender, as in the upper thoracic spine, because they will break off if the points of the jack do not embed themselves. If the scoliosis is associated with kyphosis, as is usual in the mid-thoracic and upper thoracic regions, the sleeve of the jack will not lie flat until parts of the ribs are resected. If the kyphosis is severe the sleeve will eventually lie on the parietal pleura.

Before the jack is placed, strong traction is applied to the head and legs so that as long a sleeve as possible can be used. As the sleeve is turned and the jack expands the points will embed themselves and obtain a firm anchorage. Further turning then corrects the lateral curvature. This proceeds slowly with short pauses between the turns; it takes fifteen to twenty minutes, and is stopped when the resistance to further turning suggests that the limit

FIG. 4

FIG. 5

Case 7—Congenital scoliosis. Age fifteen. Figure 4—Before correction. Figure 5—After correction.

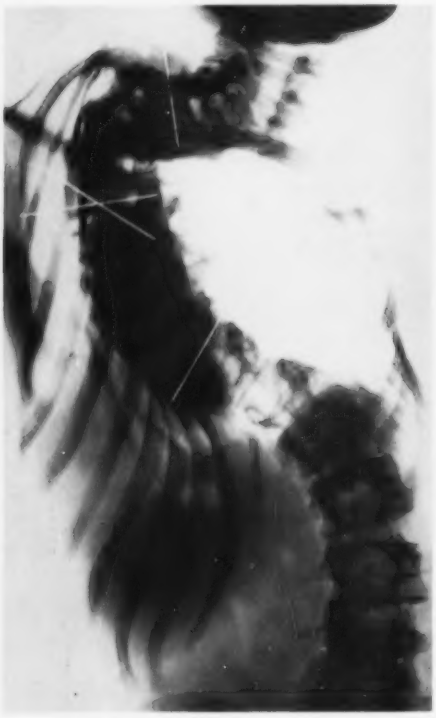

FIG. 6

FIG. 7

Case 2—Paralytic scoliosis. Age sixteen. Figure 6—Before correction. Figure 7—After correction.

of safe correction has been reached. The bed for the grafts can then be prepared and filled and the skin closed.

The operation can be completed in one stage, especially if bank bone is to hand; but proceeding by stages has definite advantages. At the first stage the jack can be inserted and opened out, and the bone grafts laid on the convex side; at the second stage, a week or two later, it will be found that the jack has loosened a little. The slack is taken up, and a further

TABLE I

DETAILS OF ELEVEN CASES OF SCOLIOSIS IN WHICH OPERATIVE CORRECTION WAS UNDERTAKEN

Case	Date of operation	Age at operation	Type of scoliosis	Extent of curve	Measurement		
					Before operation	After operation	Correction gained
PRIMARY CURVES							
1	1.11.51	12	Congenital	T.3–T.9	84°	78°	6°
2	10.3.52	16	Paralytic	T.2–T.10	140°	80°	60°
3	21.4.53	15	Idiopathic	T.9–L.2	100°	71°	29°
4	22.10.52	19	Idiopathic	T.7–L.1	99°	60°	39°
5	17.4.53	15	Congenital	T.4–T.12	92°	72°	20°
6	29.6.53	14	Paralytic	T.2–T.8	74°	56°	18°
7	3.7.53	15	Congenital	T.9–T.12	121°	83°	38°
8	12.12.53	24	Idiopathic	T.6–T.12	90°	81°	9°
9	12.1.54	18	Idiopathic	T.7–T.12	97°	67°	30°
SECONDARY CURVES							
10	14.11.52	14	Paralytic	T.12–S.1	99°	80°	19°
11	16.11.53	14	Paralytic	L.4–S.1	25°	7°	18°

correction of 10 to 15 degrees may be possible, because during the interval the discs and ligamentous attachments which resisted correction at the first stage will have relaxed or stretched; grafts are then laid in the concavity of the curve.

POST-OPERATIVE TREATMENT

No external fixation is needed after operation; if it is known that the jack has a very firm hold the patient may be encouraged to sit up in a few days' time and be got up soon afterwards. If the hold is doubtful then he can lie in a plaster shell for a few weeks to allow consolidation to begin. The jack should be left in for at least six months—in fact it might be better left indefinitely; its removal is difficult as the ends at least are embedded in the new bone.

CLINICAL OBSERVATIONS

The improvement gained in the eleven curves for which this operation has been undertaken is shown in Table I. Illustrative radiographs are shown in Figures 2 to 7.

The patients chosen for this operation all had severe deformities, and one had failed to

respond to wedging in a Risser jacket. The congenital cases showed gross structural changes and no correction occurred on suspension. One was associated with a Fallot's tetralogy which prevented any conservative treatment; and, even after Blalock's operation, the patient remained a bad subject for operation; so this was done in two stages. In two cases of severe paralytic scoliosis the lower secondary curve has been reduced and stabilised before beginning the correction of the major curve.

The complication most to be feared is pneumothorax; it increased the interval between the two stages in one case in the present short series, but should be quite easy to avoid. Two patients developed symptoms of mild ileus two or three days after operation, but it is uncertain whether the spinal correction was the cause.

COMMENT

The advantages of the method are: 1) the duration of treatment is shortened; 2) plaster jackets before and after operation are avoided; 3) the essential part of the curve is corrected; 4) curves in parts of the spine where correction is difficult can be tackled—for instance, those in the upper half of the thoracic spine; 5) curves in patients who would not tolerate plaster jackets can be corrected.

The limitations of the method are: 1) only a partial correction of a curve is possible, because the straighter the spine becomes the less efficient is the jack; major curves of less than 70 degrees should be treated by other means; 2) correction of rotation is less likely to be achieved than it is in a plaster jacket.

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WEDGE RESECTION FOR SCOLIOSIS

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The history of the treatment of scoliosis is largely one of extravagant claims which later experience has failed to substantiate. It is, therefore, with some diffidence that I put forward a new operation for the correction of this deformity. This paper is, unfortunately, no contribution to the far more important problems of etiology and prevention; it claims only to describe a good way of correcting severe, progressive uncompensated scoliosis.

Until recently the most popular way of trying to correct scoliosis has been by some form of transection jacket (Risser 1948). Although considerable cosmetic improvement is often

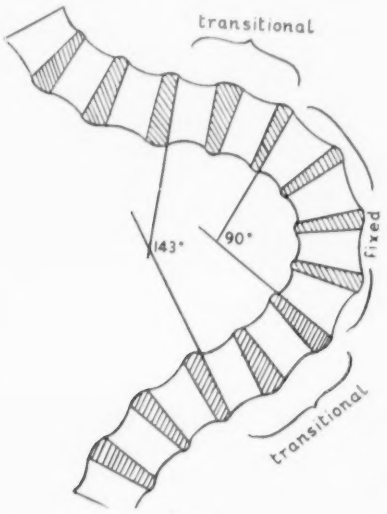

FIG. 1

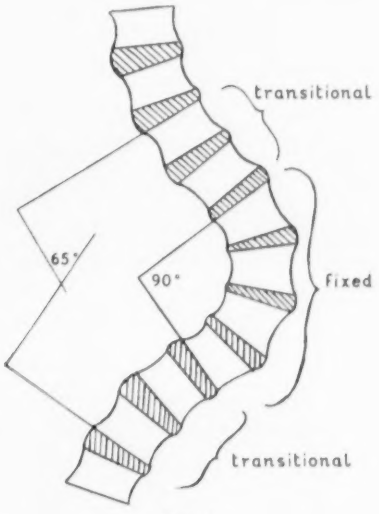

FIG. 2

Figure 1—Tracing of curve before "correction" by Risser jacket. Figure 2—After "correction" by Risser jacket. There is apparent correction from 143 degrees to 65 degrees, but the real curve remains fixed at 90 degrees.

obtained, the correction is usually more apparent than real in so far as the mobile transitional parts of the curve are straightened but the central fixed portion is unaffected (Figs. 1 and 2). Obviously the rational approach to the problem is to excise a wedge of bone from the apex of the curve—much as in the correction of a pes cavus. The operation described here differs considerably from excision of a hemivertebra (Royle 1928, Lackum and Smith 1933, Wiles 1951). It can be used both in the lumbar and in the thoracic regions. It is rational in that it achieves correction in the fixed part of the curve by a direct attack on its apex. The operation itself is relatively straightforward—certainly no more severe than the average antero-lateral decompression for Pott's paraplegia. It has been done sixteen times without any special hazards or complications, and on occasions correction of 90 degrees has been obtained. Illustrative radiographs are shown in Figures 3 to 10.

TECHNIQUE OF OPERATION

The essence of the operation consists in making an incision over the transverse processes on the convex side of the apex of the curve. The scapular muscles and longitudinal spinal muscles are split longitudinally; the deep oblique spinal muscles are detached from the

FIG. 3

FIG. 4

Case 1. Figure 3—Before operation. Curve 64 degrees. Figure 4—After wedge resection. Curve 18 degrees.

FIG. 5

FIG. 6

Case 2. Figure 5—Before operation. Curve 117 degrees. Figure 6—After wedge resection. Curve 50 degrees.

FIG. 7

FIG. 8

Case 3. Figure 7—Before operation. Curve 68 degrees. Figure 8—After wedge resection. Curve 10 degrees.

FIG. 9

FIG. 10

Case 4. Figure 9—Before operation. Curve 116 degrees. Figure 10—After wedge resection. Curve 70 degrees.

laminae, transverse processes and the inner ends of the ribs. The two laminae, transverse processes and the medial ends of two ribs are exposed (Fig. 11). The ligaments holding the medial ends of the ribs are divided and the medial three inches of two ribs are removed. The intercostal nerves are identified. The corresponding pedicles and laminae are nibbled

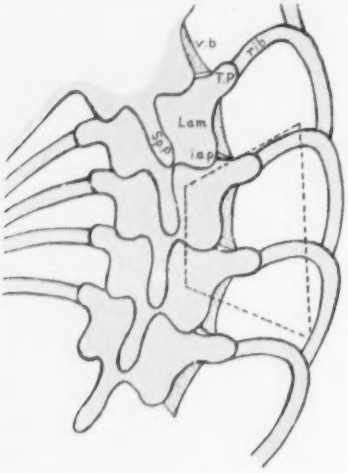

FIG. 11

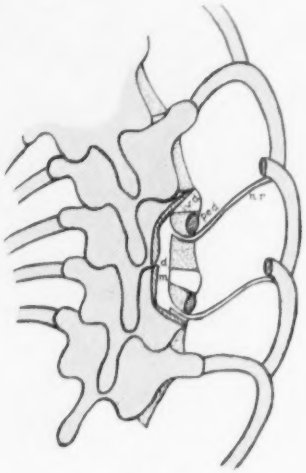

FIG. 12

Figure 11—First stage of operation. v.b.—Vertebral body. T.P.—Transverse process. Sp.P—Spinous process. Lam.—Lamina. i.a.p.—Inferior articular process. Figure 12—Second stage of operation. ped.—Pedicle. d.m.—Dura mater. n.r.—Nerve root. i.v.d.—Intervertebral disc.

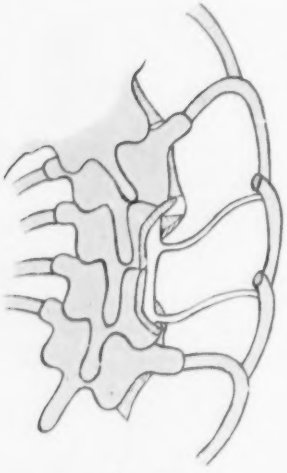

FIG. 13

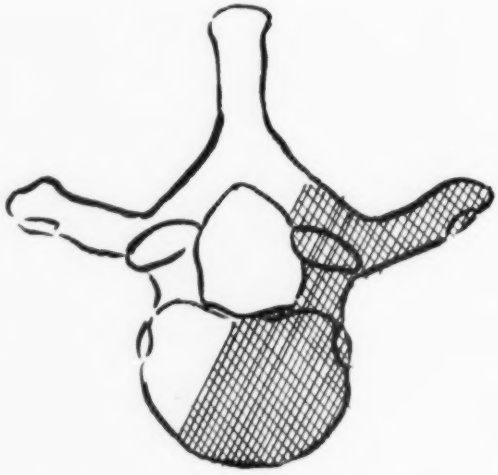

FIG. 14

Figure 13—Third stage of operation. Vertebral wedge removed. Figure 14—Showing the amount of vertebra removed.

away, the dura is exposed, and the vertebral bodies are well displayed as they are rotated towards the surgeon (Fig. 12). With a small gouge or burr and nibbling forceps the intervertebral disc space and adjacent wedges from the vertebral bodies are excised (Figs. 13 to 15). The resulting cavity is packed with a haemostatic agent and the wound is closed.

A plaster jacket including the arm and leg on the concave side is applied, securing as much correction as possible: the use of an Abbott's frame is helpful. When the plaster is dry it is wedged open by stages until maximal correction is obtained.

Recently, at the suggestion of Mr H. J. Richards, we have been using a transverse incision which appears to give better access than the original longitudinal incision. Also, in some of the older patients, we have removed more bone including a spinous process and part of the opposite lamina. Although this makes correction easier there is a risk of making the spine too unstable and it is too early to say whether or not this extended operation is an improvement.

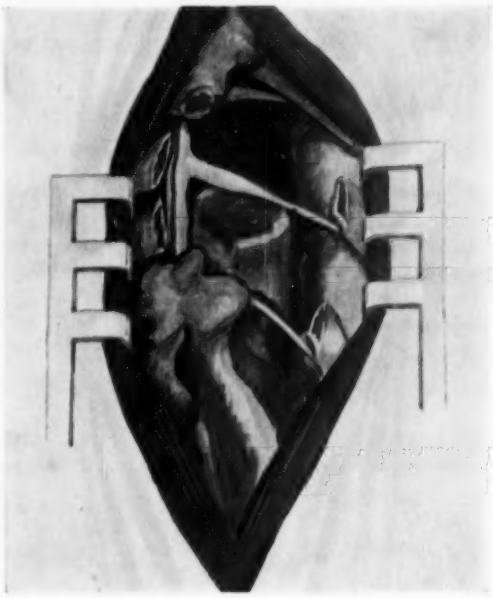

FIG. 15

The operation completed.

COMMENT

Although the operation itself is not unduly difficult it requires a very competent anaesthetist. The corrective jacket must be fitted with great care and precision. The necessary correction must be obtained within a reasonably short time, and then the spine is held in the corrected position until the partly resected vertebral bodies have fused together.

SUMMARY

" Wedge excision " of the apex of the curve is the rational way of correcting a scoliosis. It is a straightforward procedure which is successful in practice.

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TUBERCULOSIS OF THE SUBDELTOID BURSA

A Review, with a Report of Two Cases

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Tuberculosis of the shoulder joint represents less than 1 per cent of all bone and joint tuberculosis, and affection of the subdeltoid bursa is still less common. It receives scant, if indeed any, mention in current British and American orthopaedic text-books, and since it is possible that the occasional case which does occur may be incorrectly diagnosed in the earlier stages, when treatment should be most effective, a brief review of the condition is presented, together with a report of two cases.

The previous literature was reviewed by Cone when he reported a proven case in 1911. He considered that Acrel's description in 1779 of the " Ganglion Crepitans Acrellii " containing rice bodies was one of the earliest accounts of tuberculous bursitis, but in fact neither this account nor the descriptions of Cruveilhier in 1816 and of Dupuytren in 1839 of " cysts " containing rice bodies can be considered as proven cases. At that time the true pathology of the condition was not understood, and tuberculous, syphilitic and simple non-specific chronic bursitis were probably not clearly differentiated. For instance, Dupuytren thought that the cysts he described were hydatidiform cysts and that the rice bodies were parasites.

The fibrinous nature of the rice bodies was first demonstrated in 1856 by Virchow, who began the controversy on the mode of their formation. The discovery of the tubercle bacillus and the subsequent clarification of the pathology made it possible to differentiate tuberculous bursitis with certainty. It is now generally accepted that rice-body formation is not peculiar to tuberculous bursitis.

REVIEW OF PREVIOUS CASE REPORTS

Reports of tuberculosis of the subdeltoid bursa in English are few, and Cone's case reported in 1911, as already mentioned, appears to be the earliest report of a proven case. The patient was a man of twenty-two who complained of morning stiffness in the right shoulder for three years and of lancinating pain for three months. The pain had become continuous and forced him to give up work. On examination there was fullness of the shoulder, with crepitus in front of the joint and tenderness localised to the acromial region. Full abduction and medial rotation of the arm were prevented by pain. Radiographs showed rarefaction of the greater tuberosity of the humerus and roughening there and at the upper part of the glenoid fossa. There was tuberculous involvement of the apices of both lungs. At operation the bursa was found to be a thick grey sac containing 400 multi-faceted bodies; it was excised completely and the soft tip of the acromion was removed; the bone elsewhere was found to be normal. The shoulder joint was opened for inspection and found to be normal. Subsequently a sinus developed, but it healed and the patient returned to work.

In 1935 Deacon reported a presumptive case with bilateral involvement. The symptoms were of six years' duration on the right side and of four and a half years' on the left side. The patient, a man of thirty-five, complained of swelling and occasional aching, but there was no limitation of movement. Both bursae were excised and the diagnosis appears to have been based on the presence of rice bodies alone.

In 1942 Etchegorry and Blanco reported a case involving the right shoulder of a fifty-two years old man who had also a bilateral tuberculous pulmonary lesion. There was a two years' history of pain and swelling of the right shoulder which was improved by aspiration. The patient died of his pulmonary tuberculosis and the diagnosis was confirmed at necropsy.

In 1950 Kenin could find only seven cases in the records of the Hospital for Joint Diseases, New York, in the previous twenty-eight years. Only three were proven cases: in a girl of seventeen years, a woman of forty-eight and a man of thirty-eight. In all three cases the bursa was excised successfully and the diagnosis was confirmed histologically.

CASE REPORTS

Case 1—Woman aged twenty-four, married. Tapestry designer. Right handed. No personal or family history of tuberculosis.

At the age of twenty she complained of limitation of movement and swelling of the right shoulder which had become progressively worse during the previous two years. She remembered no injury to her shoulder, but attributed her condition to rowing. She was recorded as a healthy girl with a spherical swelling over the front of the right shoulder under the deltoid. Passive shoulder movements were full except for slight limitation of lateral rotation and abduction. Subdeltoid bursitis with fibrinous loose bodies was diagnosed.

FIG. 1

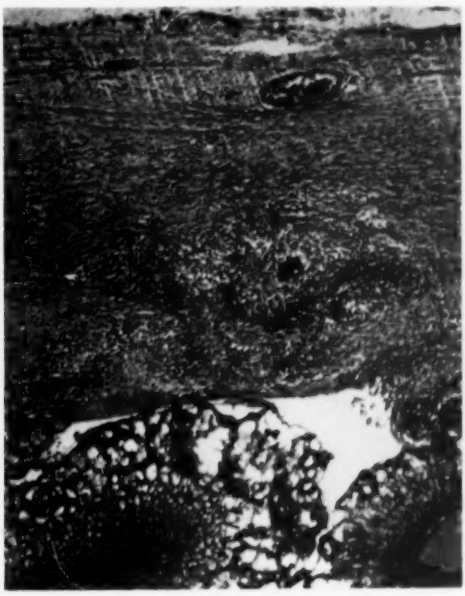

FIG. 2

Case 1. Figure 1—Radiograph of the right shoulder showing irregularity of the under surface of the acromion process and seemingly a cystic change in the greater tuberosity of the humerus. Figure 2—Photomicrograph showing the wall of the excised bursa. An epithelioid system with giant cells is seen, and the formation of two fibrinoid rice bodies is well shown.

A month later the bursa was completely excised. It contained several hundred fibrinous loose bodies which appeared to be budding from a proliferative area over the greater tuberosity of the humerus; the remainder of the bursal lining was merely corrugated. Seemingly no bacteriological or histological examination was made. After a week with the arm on an abduction frame and a short course of physiotherapy as an out-patient she was discharged two months after operation with a full range of shoulder movement.

Four years later the right shoulder again became swollen and she noticed increasing pain and a feeling of stiffness. Her general health was excellent and she allowed six months to pass before seeking advice. The erythrocyte sedimentation rate (Westergren) was 38 millimetres in the first hour. A provisional diagnosis of "tuberculous shoulder" was made and she was provided with an abduction frame while awaiting admission to hospital. Aspiration yielded 20 millilitres of opalescent yellowish fluid, but the tubercle bacillus was neither seen nor cultured.

On admission to the Wingfield-Morris Orthopaedic Hospital the patient's general nutrition and condition were good. No abnormality was detected in the respiratory, cardio-vascular or

central nervous system. The movements of the right shoulder joint were painless and limited by only 10 or 15 degrees at the extreme range in each direction. There was a large subdeltoid swelling with an extension herniating through an apparent gap in the anterior fibres of the deltoid.

Investigations—The Mantoux reaction (1/10,000) was strongly positive. The Wassermann and Kahn tests were negative. The erythrocyte sedimentation rate was 13 millimetres in one hour (Westergren). Haemoglobin was 100 per cent (14·8 grammes per cent). The white cell count and blood film were within normal limits. *Urine*—No abnormality was detected. Culture for M. tuberculosis was negative.

Radiographic examination—The chest showed multiple foci, but there was no evidence of recent spread and no definite evidence of activity. The right shoulder showed gross irregularity of the acromion and of the underlying greater tuberosity (Fig. 1).

Tuberculosis of the subdeltoid bursa was considered to be a possible diagnosis and it was decided that the bursa should be excised.

Operation—Through the previous " sabre-cut " scar the deltoid was split, revealing a large adherent bursa which was completely excised and found to be full of loose bodies. The capsule of the shoulder joint and the greater tuberosity of the humerus appeared normal.

Histological examination—The specimen was cystic and filled with rice bodies. It consisted of fibroblastic tissue surrounding a cystic cavity. There were numerous epithelioid systems in the cyst wall, with many giant cells. The numerous rice bodies were composed of fibrinoid material; some were attached to the cyst wall, but others were free within the cyst. There was a chronic inflammatory reaction around the epithelioid systems. The histological structure was morphologically that of tuberculosis (Fig. 2). Though no acid-fast bacilli were seen in sections stained by the Ziehl-Neilsen method, fluid from the cyst grew M. tuberculosis on culture.

Progress—Sterile collections of blood-stained fluid were aspirated twice after the operation. The patient received a three months' course of chemotherapy in the form of streptomycin (1 gramme daily) and isonicotinic acid hydrazide (50 milligrams three times daily). She remained in hospital wearing an abduction splint for six weeks. Thereafter she wore a collar-and-cuff sling and continued chemotherapy at home. Four months after operation the sling was discarded.

Twelve months after operation there had been no recurrence of swelling of the right shoulder. The deltoid muscle had regained its normal power and shoulder movements lacked only about 5 degrees of the full range. Chest and shoulder radiographs showed no activity of the disease, and the erythrocyte sedimentation rate was 4 millimetres in 1 hour (Westergren).

Case 2—Man aged thirty-six, single. Electrician. Right-handed. No family history of tuberculosis.

This patient strained his right shoulder while strap-hanging on the London Underground. During the ensuing month he noticed stiffness, pain and some swelling in the shoulder. When seen at a hospital six months later he was found to have subdeltoid bursitis, probably with rice bodies. Radiographs showed some rarefaction in the region of the greater tuberosity. Operation was declined by the patient who received physiotherapy with little benefit.

Two years later the pain increased and at a second hospital tuberculous subdeltoid bursitis was diagnosed and was confirmed by aspiration. Radiographs showed tuberculosis of the lungs but there was neither clinical nor radiological evidence that the shoulder joint itself was infected. Treatment was by rest in bed for three months, with a full course of streptomycin and para-amino-salicylic acid. This was followed by three months' convalescence.

When the patient was first seen at the Wingfield-Morris Orthopaedic Hospital in the following year there was fullness of the subdeltoid region and a distinct fluctuant swelling along the line of the long head of biceps (Fig. 3). The range of shoulder movements was limited by only a few degrees at the extremes of each movement. Aspiration of the bursa yielded a small quantity of yellowish pus, thick with fibrinous flakes. On direct staining no organisms were seen, but tubercle bacilli were grown on culture. An attempt was made to relieve the condition by aspiration and streptomycin replacement, but without success.

Investigations—The Wassermann and Kahn tests were negative. The erythrocyte sedimentation rate was 3 millimetres in one hour (Westergren). Haemoglobin was 103 per cent (15 grammes per cent). The white cell count and blood film were within normal limits. *Urine*—No abnormality was detected. Culture for M. tuberculosis was negative. Agglutination tests with S. typhoid and parathyroid suggested previous immunisation. Agglutination tests with the brucella group were negative; tubercle bacilli were not found in three specimens of gastric washings.

Radiographic examination—A lesion of the outer end of the acromion shown in the early films now appeared to be partly healed. A cavity in the acromion had for the most part become filled in (Fig. 4). An arthrogram of the shoulder showed no alteration from the normal, and there was no communication with the subdeltoid bursa. Chest radiographs showed apical and sub-apical infiltration on both sides, with evidence of fibrosis and shrinkage over a year.

Operation—Through a " sabre-cut " incision the deltoid muscle was split. Under the deltoid there lay fibro-fatty tissue with no evidence of a residual cavity except at its distal limit, where there was a small dumb-bell shaped cavity containing a little pus, a few flakes of fibrin and some watery fluid. The diseased tissue was excised completely. The acromion process and the greater tuberosity appeared normal. The shoulder joint itself was not explored. One gramme of streptomycin powder was inserted locally.

Histological examination—Sections showed connective tissue with considerable chronic inflammatory reaction and numerous clefts lined by poorly vascularised chronic inflammatory granulation tissue of tuberculous type. Epithelioid systems were fairly frequent and giant cells numerous. Large amounts of fibrinoid caseous material were present within the clefts. The appearances were morphologically those of tuberculous bursitis.

Progress—A three months' course of chemotherapy (streptomycin 1 gramme daily and isonicotinic acid hydrazide 50 milligrams three times daily) was given. The wound healed by first intention. An abduction splint was worn for one month after operation and then gradually discarded in favour of a collar-and-cuff sling. Two months after operation the patient was discharged to a convalescent home.

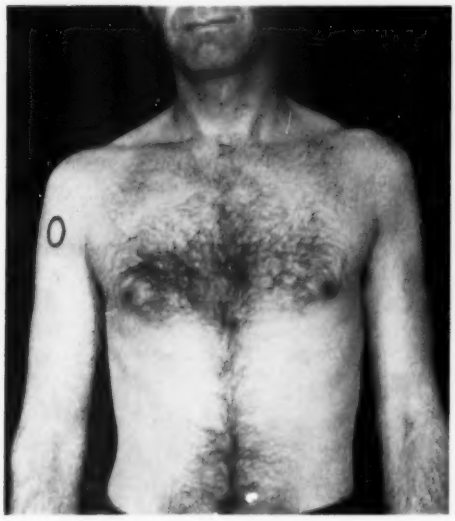

FIG. 3

FIG. 4

Case 2. Figure 3—Photograph showing site of the extension from the subdeltoid bursa, where a residual active focus of tuberculosis was demonstrated. Figure 4—Radiograph of the right shoulder. The acromion process shows irregularity like that shown in Figure 1.

Nine months after operation the patient suffered no discomfort from the shoulder and was in excellent health. Shoulder movements were restored to the pre-operative range and there was good deltoid power. The chest lesion had remained stable.

DISCUSSION

Tuberculosis of the subdeltoid bursa, although rare, is a distinct entity, and if the diagnosis is borne in mind it should be possible to differentiate it from non specific bursitis on the basis of the symptoms, clinical examination and radiographic pictures. The condition is of insidious onset and a year or more may pass before advice is sought. Pain does not appear to be a prominent feature in the early stages, and is often more in the nature of a feeling of stiffness. Constitutional signs are absent unless there is an active tuberculous focus elsewhere in the body. There is local swelling of the shoulder, associated in some instances with crepitation, tenderness and increased warmth. Limitation of movement, although reported, was not pronounced in the two cases described here, and it seemed

mechanical in origin. Diagnostic aspiration is useful because isolation of the tubercle bacillus allows chemotherapy to be begun before operation.

In these cases radiographic examination showed changes in both the acromion and the greater tuberosity of the humerus, like those reported by Cone (1911) and by Kenin (1946, 1950). At operation the bone appeared healthy and, as pointed out by Kenin (1946), such changes probably represent rarefaction from local hyperaemia rather than tuberculous involvement of the bone.

The treatment of choice is radical excision of the bursa if the patient's general condition allows. Ideally, streptomycin combined with one or more of the other available anti-tuberculous drugs should be begun before exploration. Jefferson *et al.* (1950) reported excellent results from a two weeks' course of streptomycin after excision of tuberculous greater trochanteric bursae. However, in our cases a full course of streptomycin (total dose 90 grammes) was favoured.

SUMMARY

1. The previous literature in English on tuberculosis of the subdeltoid bursa is reviewed.
2. Two further cases are reported.
3. Emphasis is placed on the neglect of the condition in standard text-books and the need for bearing the diagnosis in mind.
4. Treatment by radical excision, a period of immobilisation and full supportive antibiotic therapy are recommended.

I wish to thank Dr F. H. Kemp for the radiological reports and Dr R. A. Sladden for the histological reports. I would like to express my indebtedness to Professor J. Trueta, under whose care the two patients were, both for permission to publish this article and for his help and encouragement in preparing it.

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STRESS FACTOR IN THE DISC SYNDROME

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The complexity of the disc syndrome, and the frequency with which we meet it, is perhaps sufficient to justify a slightly unusual approach, and the presentation of a sequence of ideas supported by experimental evidence.

Consider first the sequence of events on the clinical side, which has been—in part at least—responsible for the development of the idea. 1) There is an impression among many of those conducting large clinics that an increased number of people have suffered from the disc syndrome during and since the recent war, with all the symptoms of acute and chronic back pain and root irritation. Statistics relating to the frequency of the conditions are unreliable, because of the popularity of the diagnosis, but any figures produced would strongly support this contention. 2) In general, conditions that have shown a marked increase in the years during and since the war are the virus diseases and the psychosomatic disorders. Perhaps because of this, and perhaps as the last line of defence, there is an increase in the emphasis on the psychological aspect of the problem in the disc syndrome. For example, Barr (1951) has stated: "Low back and sciatic pain signify a state of stimulation of the cerebral cortex, and are not an affection of the back: it is important to study and treat the patient and not just his back." 3) Injury to the back was at first thought to be a direct and frequent causative factor. More recently less importance has been attached to it. Attacks of acute back pain have been known to come on after exposure, strenuous exercise without back injury, and prolonged emotional strain. It seems likely that these influences would be more likely to produce their effect through some general physiological reaction, rather than as a result of any direct effect on the back. 4) There is little doubt that the understanding of the syndrome is inadequate, and the treatment therefore often haphazard and empirical. None of the accepted forms of conservative treatment is universally successful in dealing even with the acute attack expeditiously, let alone having any influence in preventing subsequent attacks. Most of the ardent advocates of surgical treatment are now prepared to admit that it has a limited place in the treatment of the syndrome.

To summarise the points in the clinical hypothesis: The incidence of the condition has greatly increased, as has the emphasis on the psychological or functional aspect of the problem. Attacks have been reported after incidents in which the back was not directly involved, but in which a general psychological reaction had certainly occurred. Accepted methods of treatment leave much to be desired.

It is suggested that a sequence of events occurs in which the pressure within the annulus increases beyond the normal level (in some instances leading to acute back pain) and that this may be repeated until rupture of the annulus occurs in those discs naturally subjected to the greatest pressure. It is further suggested that emotional strain may be a factor in producing the periodic increases in pressure.

There is one significant type of radiographic appearance which may accompany the early attacks of acute back pain. The disc, instead of being its normal shape or narrowed, is expanded—particularly at the centre—and the opposing surfaces of vertebral bodies above and below are rounded to accommodate it (Fig. 1). This change is certainly not a constant finding, but it is one that merits more attention than it has received. The widened disc is associated with the acute attack of back pain, but when root signs have been present for any length of time it is no longer present—though even when the disc is narrowed the

"scalloped" vertebral bodies may give evidence of the previous existence of the state of affairs described above.

There is nothing new about the suggestion of altering tension within the disc. Hirsch (1951) made an exhaustive study of the subject. He pointed out that increasing the pressure by injection into the disc reproduces the typical pain; and he found that the changes in the nucleus precede those in the annulus, which habitually ruptures from within outwards.

It has been shown that the disc can take up large quantities of fluid when placed in solutions of low osmotic pressure (Charnley 1951); and that by placing a preparation of fresh post-mortem material, consisting of an intact disc and two vertebral plates, in water or normal saline a very high pressure can be produced within the disc (Naylor and Smare 1953).

It may seem that the above observations bear little relationship to the conditions existing in life, but reference to the work of Opie and Rothbard (1953) suggests that this is not so.

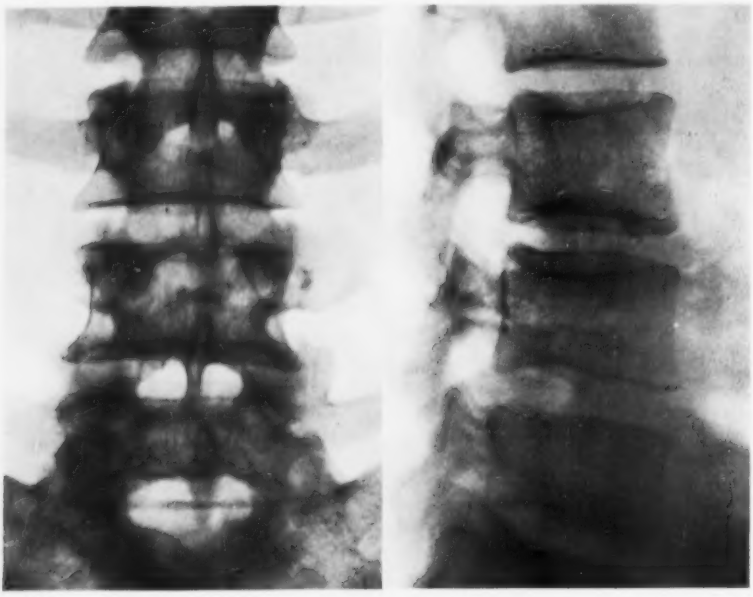

FIG. 1

Central expansion of disc suggested by hollowing of opposing surfaces of vertebral bodies.

The reaction of collagenous tissue exposed to solutions of varying osmotic pressure has been extensively studied, and Opie and Rothbard wrote: "It is noteworthy that dense fibrous tissue, which in view of its structure seems little pervious to the movement of water, is peculiarly adapted to absorb and hold it." It also seems that collagenous tissue, which contains a considerable quantity of mucopolysaccharide, is capable of taking up even larger quantities of fluid. It is somewhat uncertain whether this process is one of osmosis or hydration. There is therefore some evidence that the disc is capable of taking up fluid, that this leads to an increased pressure within the annulus, and that increased pressure leads to pain.

Disturbance of the water balance leading to retention may come about in various ways. Sudden and prolonged emotional strain leads to an increased secretion of vasopressin, the antidiuretic hormone of the pituitary. Adrenalectomy, and the interference with secretion of the adrenal, increases the antidiuretic effect of various systemic pressor substances. In physical trauma (including surgery), sometimes of even a minor degree, there is an increase

in adrenocortical activity—also an associated alteration in the water balance and retention of electrolytes, particularly sodium and chloride (Le Quesne 1953). It is known also that the administration of A.C.T.H. or cortisone causes a retention of salt and water. There is no reliable evidence as to how this retained fluid is distributed.

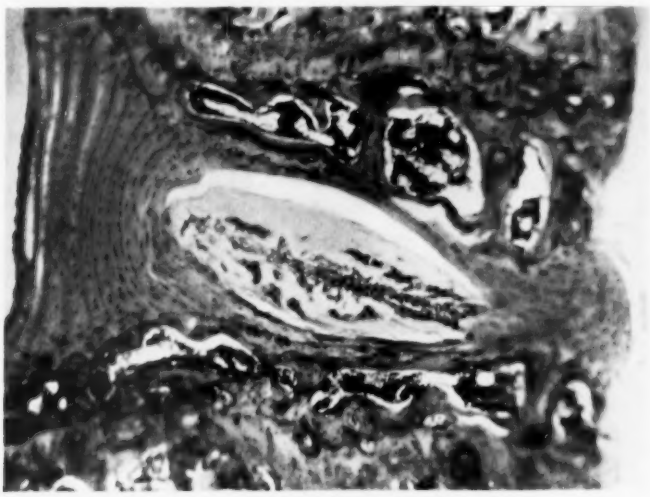

FIG. 2

FIG. 3

Typical appearance of disc from control animal (Fig. 2) and experimental animal (Fig. 3).

EXPERIMENTAL METHOD AND RESULTS

With these things in mind it was decided to investigate, in animals, the effect of stress on the intervertebral disc and the size of the nucleus.

The response of the body to non-specific stress has been studied by many people. Certain changes found in animals in response to stress, and considered to be characteristic of the

" general adaptation syndrome " described by Selye (1950), include involution of the thymus and enlargement of the adrenals. There seems to be a fairly wide measure of agreement that these changes are evidence of the reaction of the body to stress which may be applied in different ways.

It has been shown (Clarke 1953) that when voles (*Microtus agrestis*) have been subjected to daily periods of fighting their adrenals increase in weight, the thymus decreases, and the spleen shows a pathological change. In Clarke's experiments the stressor agent was a pair of old voles into whose cage a young animal had been introduced. Organ weights of these young animals were compared with those of littermate controls.

Preliminary tests were carried out at the Bureau of Animal Population, Oxford, by Dr D. H. Chitty, using methods similar to Clarke's, but under less rigidly controlled conditions; for example, the control and experimental animals were not all littermates, but had been selected at random from the males available at the time. These tests will not be described in detail; it need only be said that the experimental voles were in contact with strangers for about two hours on each of three or four days, after which they and their controls were killed. Dr A. H. T. Robb-Smith was kind enough to examine the spines, and a summary of his report is as follows. The specimens consisted of vertical sections of the vertebral columns of eighteen voles—eight control and ten experimental animals. There were no gross histological differences between the two series, although one gained the impression that in the experimental animals the nucleus pulposus showed a greater basophilia. Measurements were made to find out if there was any alteration in the volume of the nucleus pulposus, by determining the ratio between the total disc area and the area of the nucleus pulposus. Seventy-nine discs were measured, and it was found that in the control series the mean of the ratios of the disc area to nucleus pulposus area was 2·2304, with a range from 1·62 to 3·50; in the experimental series the mean was 1·9041, with a range from 1·35 to 3·35. Dr Rosemary Biggs examined these figures statistically and found that the difference of the means of the two series was 0·3263. By the " t " test, " t " = 3·0256, which is highly significant, the probability of obtaining this value by chance being between 1 : 100 and 1 : 1,000. Thus it appeared that in the experimental animals the area of the nucleus pulposus in relation to the total disc area was larger than in the control animals, the difference being statistically significant.

Because of the encouraging results of these preliminary experiments it was decided to repeat the experiment using only littermates, and excluding all possible sources of error. The size of nucleus relative to the total area of the intervertebral disc was estimated by the same method as that used in the previous studies. One hundred and thirty-one discs in twenty-four spines were measured, the measurer having no knowledge as to which were controls and which experimental animals (Figs. 2 and 3). Statistical analysis of these measurements showed a highly significant increase in the relative size of the nucleus proportional to the changes in adrenals, thymus and spleen. The details of these experiments and their results are being prepared for publication. They provide evidence that the reaction to " stress " which leads to certain changes in adrenals, thymus and spleen—thought to be due to increased adrenocortical activity—also leads to an increase in the size of the nucleus pulposus (Chitty *et al.* (MS.)).

COMMENT

It is obvious that these results do no more than add a little corroboration to an idea which has developed as a result of clinical observation. They do not of themselves make any direct contribution, but it is just possible that they point in a direction which may be of some value in relation to etiology and treatment, and which has not so far been adequately investigated.

I should like to acknowledge the valuable assistance of Dr A. H. T. Robb-Smith in the preparation of this paper, and to thank Dr Rosemary Biggs for the statistical analysis of the first series.

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INTRA-ARTERIAL TRANSFUSION IN THE TREATMENT OF SEVERE INJURY

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This paper is a description of a method of intra-arterial blood transfusion using oxygen under pressure. An account is given of its use in three patients in severe shock after multiple injuries. It has also been used in seven patients during operations in which severe loss of blood was possible.

Intra-arterial transfusion of blood was suggested by Halsted (1883) as a treatment of carbon monoxide poisoning. Much of the published experimental work was done in America towards the end of the first half of this century. In experiments on dogs Kohlstaedt and Page (1943) found that intra-arterial transfusion was superior to the intravenous route in the

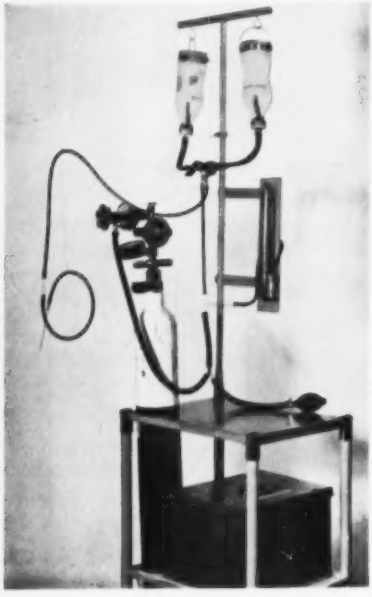

FIG. 1
Photograph of the apparatus assembled.

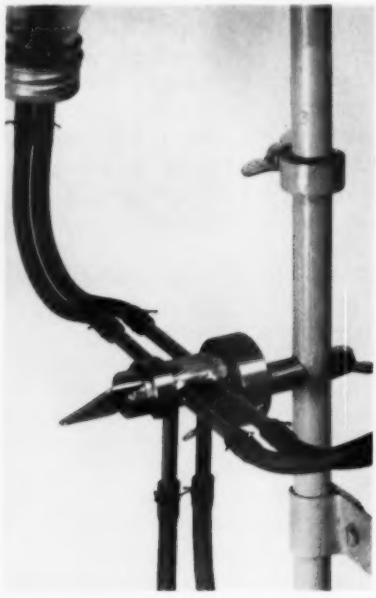

FIG. 2
Detail of the double two-way tap.

treatment of severe haemorrhage, especially when the volume transfused was less than the volume lost. Porter, Sanders and Lockwood (1948) described a method of intra-arterial transfusion in man using oxygen under pressure. The method described here is an adaptation of that method.

Many other methods using machine and hand pumps have been described (Robertson, Trencher and Dennis 1948; McKenzie and Morton 1951; Seeley, Nelson and Wesolowski 1952; Hamilton, Drye, Kinnaird and McGowan 1952; Brown 1953).

The advantages of intra-arterial transfusion are: 1) a rapid transfusion can be given to

shocked patients with collapsed peripheral vessels; 2) there is much less danger of overloading the pulmonary circulation, especially in the elderly patient; 3) rapid increase in blood pressure and improved tissue perfusion can be obtained; 4) it combats the harmful and sometimes lethal effects of tissue anoxia caused by prolonged hypotension; and 5) a small quantity of blood intra-arterially gives better results than the same volume intravenously (Kohlstaedt and Page 1943).

The disadvantages of this method are: 1) it requires more apparatus and more supervision; 2) it is sometimes necessary to expose and ligate an artery; and 3) severe ischaemic changes may occur in the limb distal to the site of the transfusion.

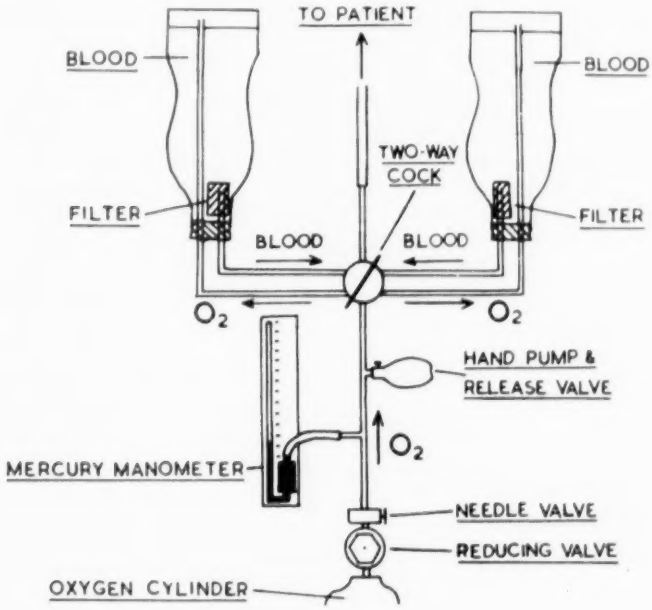

FIG. 3
Diagram of the apparatus.

Indications for intra-arterial transfusion—The indications for intra-arterial transfusion in surgery are at present: 1) severe shock after injury which is not responding to intravenous transfusion (Robertson *et al.* 1948); 2) severe blood loss especially in elderly patients—for example, after massive haematemesis when surgical intervention is contemplated; and 3) replacement of blood which has been withdrawn to induce hypotension during a major surgical procedure (Brown 1953).

TECHNIQUE OF TRANSFUSION

Using the apparatus shown in Figures 1 to 3, the transfusion is made by forcing blood from a standard blood bank bottle into an artery of the recipient by means of the pressure supplied by a cylinder of oxygen. As shown in Figure 3, the oxygen is led through a reducing valve which reduces the pressure to half an atmosphere, and then through a needle valve to regulate the flow. The oxygen then passes through a tube to which is connected a mercury manometer and a hand pump fitted with a release valve, and then on to the double two-way tap of the apparatus (Fig. 2). From the proximal part of the double two-way tap it passes to one of the bottles. The blood is forced out of the bottle through the distal part of the

two-way tap and along a length of semi-transparent plastic tubing to the patient. When the bottle is almost empty the tap is turned through 90 degrees and the flow continues without interruption from the second bottle. The first bottle can then be replaced by a full bottle while the second bottle is in use. The hand pump is used if the oxygen supply fails, and the release valve lowers the pressure should it become excessive. A simple " Record " nozzle is fitted to the end of the tubing. The needle need not be locked in position or tied to the nozzle. Long wide-bore needles are used. The apparatus is mounted on a mobile trolley. The tubing, tap, bottle attachments and needles are autoclaved and kept assembled in the theatre ready for an emergency.

The apparatus is assembled with two bottles of blood. The tubing distal to the bottles is filled with blood by gravity before the oxygen tube is attached to the cylinder.

The chosen artery is exposed and the needle inserted pointing proximally. The nozzle of the apparatus is connected to the needle and the flow of blood controlled by the pressure of the oxygen in the bottle, which in turn is controlled by the needle valve. This pressure need seldom be higher than two hundred millimetres of mercury. The pressure can be kept constant, and the changes of pressure that occur when a hand pump is used are avoided. The rate of flow can be determined from the rate of fall of the blood in the bottle.

Two arteries have been used, the radial artery at the wrist and the femoral artery in Scarpa's triangle. Any large artery may be used if it is exposed by the injury or at operation. When the radial artery was used it was ligated above and below the site of injection and divided. Firm packing of the wound was sufficient to stop bleeding from the femoral artery after withdrawal of the needle.

CHOICE OF ARTERY AND INCIDENCE OF COMPLICATIONS

Both sites have their advantages and disadvantages. The radial artery can be exposed quickly and if necessary ligated without great risk of ischaemia, and a needle can be controlled more easily in this vessel than in the deep-seated femoral artery. On the other hand, because of the difference in calibre of the vessels, the rate of flow possible in the radial artery is less than in the femoral. Ischaemic changes were more frequent when the radial artery at the wrist was used; they varied from patchy cyanosis of the skin of the forearm to transient anaesthesia and paralysis of the forearm and hand muscles. No permanent vascular complication occurred.

Several cases of gangrene of the hand complicating intra-arterial transfusion through the radial artery have been reported (Holden 1952; Porter *et al.* 1948; Yee, Westdahl and Wilson 1952). Comparison of these cases with that of the large series of uncomplicated transfusions of Brown (1953) suggests several factors in the causation of ischaemia. 1) Citrated blood stored for two weeks and injected at low temperatures tends to cause anoxia of the local tissues because both the storage (Valtis and Kennedy 1953) and the temperature (Barcroft and King 1909) reduce the amount of oxygen made available from the blood. Brown (1953) used fresh warm blood. 2) Intra-arterial injection of cold blood may cause arterial spasm (Holden 1952). 3) A duration of the transfusion exceeding two hours (Porter *et al.* 1948). 4) Injection of fluids other than blood (Brown 1953). 5) Congenital absence of an anastomosis between the radial and ulnar arteries in the hand.

CASE REPORTS

Case 1—The patient, a man aged forty-seven, sustained multiple compound fractures of both legs by the bursting of a cylinder of air at a pressure of a thousand pounds per square inch. On admission to hospital his blood pressure could not be recorded and respirations were slow and sighing. He was placed in an oxygen tent and given an intravenous transfusion of 1,000 millilitres of plasma followed by 1,000 millilitres of blood. During this time no improvement in his condition occurred. An intra-arterial transfusion of 1,000 millilitres was then given by the left radial artery in fifteen minutes (Fig. 4). During the transfusion his general condition began to improve and

the pulse quickly became palpable. At the end of the transfusion the blood pressure was 100 millimetres mercury and the patient was taking an interest in his surroundings and answering questions. During the transfusion the forearm and hand were found to be anaesthetic, the skin was cyanosed in patches, and the muscles paralysed. The condition of the limb slowly recovered to normal during the thirty minutes immediately after the transfusion.

An above-knee amputation of the right thigh and wound toilet and plaster fixation of the left were carried out initially, but a below-knee amputation of the left leg was required after two days. The patient has now returned to his previous employment, walking well with artificial limbs.

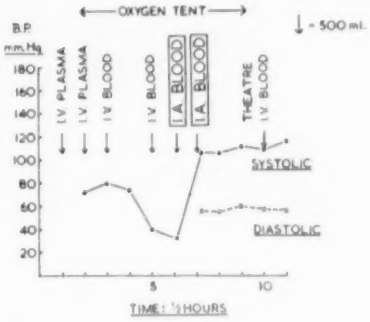

FIG. 4

Case 1—Graph of the blood pressure readings recorded during transfusion.

Case 2—Man aged thirty-two. This patient was involved in the same accident as the patient in Case 1. He sustained a traumatic amputation high in the left thigh, and a large degloving injury of the right thigh and knee. On admission his general condition was better than that of the other patient. He was given 1,000 millilitres of plasma followed by 1,000 millilitres of blood, but this did not stop a gradual deterioration of his condition. He was then given an intra-arterial transfusion of 1,000 millilitres of blood into the left radial artery with an excellent response, as shown in Figure 5. In this case patchy cyanosis of the forearm and hand was present during the transfusion. This passed off after the transfusion and no residual complication was present after twelve hours.

Suture of the stump of the left thigh and extensive split-skin grafting of the right leg were required. He is now back at work with a left artificial limb.

Both patients stated afterwards that they were conscious immediately after the accident, but did not remember any events thereafter until about the end of the intra-arterial transfusion.

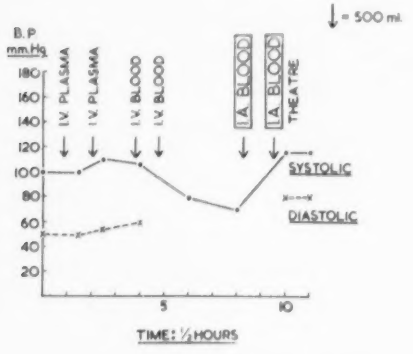

FIG. 5

Case 2—Graph of the blood pressure readings recorded during transfusion.

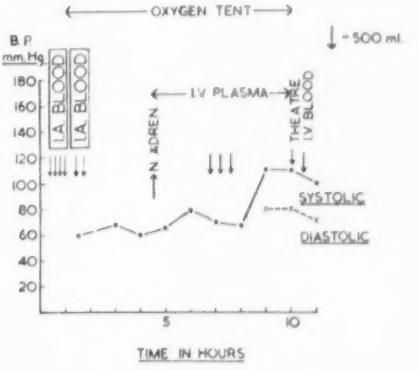

FIG. 6

Case 3—Graph of the blood pressure readings recorded during transfusion.

Case 3—A woman of fifty-six was run over by a bus, sustaining severe fractures and soft-tissue injuries of both legs. For a year before the accident her general health had been poor. On admission to hospital she was pale and conscious, but neither her pulse nor blood pressure could be obtained. Although the patient appeared to be dying, she was placed in an oxygen tent and an intra-arterial transfusion of 2,000 millilitres of blood was given into the femoral artery of the most seriously injured leg. After this the blood pressure was recorded at 60 millimetres Hg. (Fig. 6), but the pulse was very rapid. Since her general condition had not improved as much as expected the transfusion into the femoral artery was stopped and after a short interval 1,000 millilitres of blood was given through the radial artery, but without any marked improvement in her condition. It appeared that the patient was in irreversible shock. A slow intravenous drip of plasma was set up, and four and a half hours after admission two milligrams of noradrenaline was added to it.

After this the blood pressure was 80 millimetres Hg., and the pulse was stronger. The intravenous plasma drip was continued, and nine hours after admission her blood pressure was 110 millimetres Hg., and she was then fit for operative treatment.

Slight patchy cyanosis of the skin of the volar aspect of the forearm was present during, and for one hour after, transfusion, but no reaction was seen in the leg.

The left leg was amputated above the knee and split-skin grafts were applied to the right leg. She later required further skin grafts to the right leg and for many months was mentally unstable. Eighteen months after the accident she is now much improved and is beginning to walk with a left artificial limb and a right caliper.

I wish to thank Mr Roland Barnes for his assistance in the preparation of this paper and also Mr A. McDonald and Mr T. Hotham of the Technical Staff of the Western Infirmary, who were responsible for the construction of the apparatus.

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MINOR HAND INJURIES

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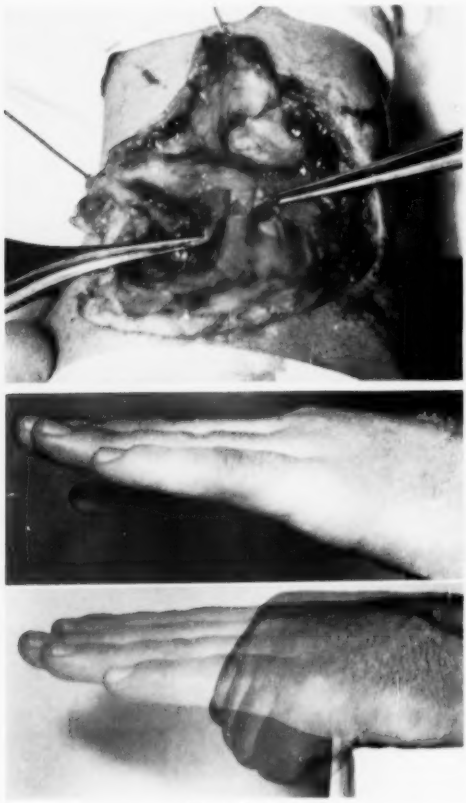
Little originality is claimed for this paper, which reports some of the cases treated at the London Hospital during the last year. There are nearly 24,000 attendances in the Orthopaedic and Accident Department each year, of which about 6,000 are accident cases. Since no beds can be reserved for these patients the vast majority have to be treated as out-patients and operated on under local anaesthesia.

Apart from domestic accidents, most of the injured hand cases come from the surrounding tailoring and wood-working industries. The title of this paper was deliberately chosen to exclude the more dramatic major mutilations. So-called " minor " injuries of the hand are responsible for an enormous loss of working time, and by laying down standard methods of treatment we have found that we are returning patients to work earlier than in previous years.



FIGS. 1-3

Extensor tendon injury at dorsum of wrist. Figure 1—At time of operation. Figure 2—Six weeks later: lag in extension of index finger. Figure 3—Final result.

With the publication of Rank and Wakefield's book (1953) stressing the principles of repair in the treatment of hand injuries, a reasonable classification and outline of treatment have become readily available. Most of the cases reported here fit into that classification, and in general the treatment has been found to follow the recommendations of Rank and Wakefield.

Classification—The cases described are classed under the headings: extensor tendon injuries, finger tip injuries, and skin flap injuries.

ILLUSTRATIVE CASES

EXTENSOR TENDON INJURIES

Rank and Wakefield rightly stressed the difficulties frequently met in trying to obtain a result which is of a high standard both from the surgical and practical viewpoints.

The cases to be described in this group illustrate injuries to the tendons at the four common sites: the dorsum of the wrist, over the metacarpo-phalangeal joint, over the proximal interphalangeal joint, and at the distal insertion of the tendon.

The dorsum of the wrist—Figure 1 shows the left hand of a youth of seventeen who fell through a window, cutting the extensor tendons to the index, middle and ring fingers.

Fortunately, the flap which had been raised was based proximally and gave a good exposure of the tendons, which were repaired with fine wire. Figure 2 shows the lag in extension of

FIG. 4

FIG. 5

Extensor tendon injury over metacarpo-phalangeal joint. Figure 4—Top picture shows initial injury; lower picture shows result. Figure 5—Range of movement.

FIG. 6

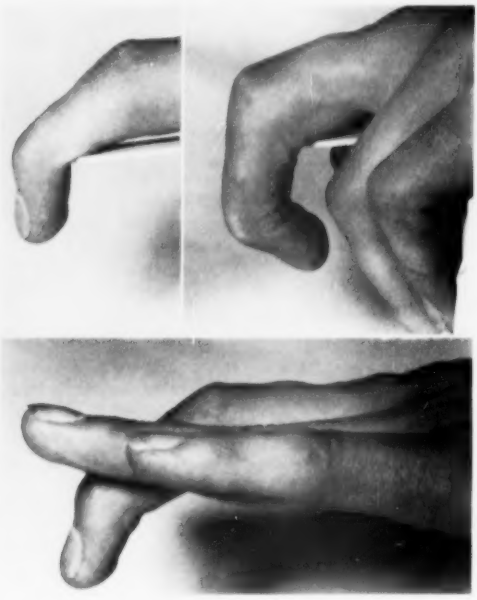


FIG. 7

Cog-wheel injury with severance of extensor tendon over proximal interphalangeal joint. Figure 6—Initial state. Figure 7—Result at six months.

the index finger six weeks after injury; with active exercises the slack was taken up within two months and the final result is shown in Figure 3.

The metacarpo-phalangeal joint—Figure 4 (top) shows the right hand of a man in which there was actual loss of skin and extensor tendon caused by a circular saw injury. The joint was not damaged. The severed tendon was sutured by fine wire and a rotation-advancement flap was moved to replace the skin loss. The bottom picture shows the result obtained five weeks after injury and Figure 5 indicates the range of movement.

The proximal interphalangeal joint—Figure 6 shows a cog wheel injury to the left middle finger of a printer. The extensor tendon was virtually severed over the proximal interphalangeal joint, the capsule of which was also penetrated. Both wounds of the tendon were sutured with fine wire and the skin wounds closed with nylon sutures. Figure 7 shows the result six months after injury. The range of movement is: proximal interphalangeal joint—active 165–115 degrees, passive 170–100 degrees; distal interphalangeal joint—active 145–100 degrees, passive 180–100 degrees. This finger illustrates the type of case that can be considered

FIG. 8

FIG. 9

Crush injury causing mallet finger. Figure 8—Initial state. Figure 9—Result.

a bad surgical result but an acceptable practical one in that the deformed finger is no hindrance to its owner.

The distal interphalangeal joint—Figure 8 shows the result of a crush injury to a woman's left ring finger. The bony fragment was removed and Figure 9 shows the very indifferent result obtained. The patient is, however, quite satisfied with the finger. The range at the joint is: active 180–160 degrees; passive 180–150 degrees.

That mallet fingers are notoriously difficult to treat satisfactorily is shown by the great variety of treatments suggested. When there is an open wound with bony damage, suture must always be tried, but we have found accurate reposition of the bony fragment often extremely difficult.

FINGER TIP INJURIES

These are by far the most common injuries treated and, after cut/crush ratio (Barron 1947) has been determined, are classified for treatment as small " tidy " areas, nail injuries, partial

FIG. 10

FIG. 11

Slice wound through nail. Figure 10—Initial state. Treatment by immediate split-skin grafting. Figure 11—Result.

and complete amputations, and pulp loss. Small areas of " tidy " epithelial loss are replaced by a thick split-skin graft and need no comment.

Nail injuries—Slicing wounds frequently involve both the nail and finger tip. These wounds should be treated by an immediate split-skin graft. Ideally, the graft should be cut thinner at one end. The thin part should be placed over the nail area and the thicker part over the finger-tip wound. Figure 10 shows the finger of a butcher which was treated in this manner. As the nail grew it covered the thin part which became incorporated in the nail bed (Fig. 11).

Partial amputation—Injuries such as that shown in Figure 12 are common in the tailoring industry, being caused by a power-driven cutting machine. Provided that one of the vascular bundles is intact, the flap, even if it includes the bone, should be sutured back into place,

FIG. 12

FIG. 13

Partial amputation. Figure 12—Initial state. Figure 13—Result after suture.

the hand kept elevated and the minimum of dressings applied. The results are almost invariably good (Fig. 13).

Complete amputation—Rank and Wakefield stressed that considerations of ideal amputation sites do not apply in the immediate treatment of traumatic amputation of digits. All available skin should be used to give the greatest length possible. Figure 14 shows a crush injury to the right little finger of a stonemason. The radiograph (Fig. 15) shows that there was extensive exposure of the distal phalanx. The phalanx was, therefore, filleted out and the remaining soft tissues were used to cover the defect. Figure 16 shows the final result.

FIG. 14

FIG. 15

FIG. 16

Complete amputation. Figure 14—Initial state. Figure 15—Radiograph showing exposure of phalanx. Figure 16—Result after filleting the phalanx and closure of soft tissues.

Pulp loss—When there has been considerable pulp loss of the " tidy " type, it is our practice to use a palmar flap. Figure 17 shows the right ring finger of a man's hand that had the pulp removed by being crushed in a car door. A palmar flap was attached for two weeks (Fig. 18); the final result is shown in Figure 19. Figure 20 shows a man's right hand injured in a machine cutter. The thumb was intact; the index finger had minor lacerations; the middle finger had considerable pulp loss; the distal phalanx and nail of the ring finger had been avulsed and a ragged flap had been raised on the palmar surface of the little finger. The middle finger was treated by a palmar flap, the ring finger by removing the phalanx and completing the amputation, and the flap on the little finger was loosely sutured into place. Figure 21 shows the final result.

We have no hesitation in recommending the use of the palmar flap for reconstituting these finger tips. Rank and Wakefield did not recommend the practice and Robins (1954) has recently described his use of the post-auricular graft in these cases. It seems probable that skin from a distance must be an inferior substitute for a full-thickness skin flap with associated fat. The palmar flap skin comes from an area which is accustomed to trauma and contains in far greater abundance than skin from any other area the appropriate nerve endings into which regeneration can take place. The operation is frequently condemned on the grounds that a second defect is created in the hand. If a thick split-skin graft is sewn into the donor site the resultant area is perfectly serviceable and the photographs show that it soon takes up the normal palmar creases.

SKIN FLAP INJURIES

The two cases to be described in this section illustrate a problem that is sometimes presented by flaps raised by injury.

Figure 22 shows a small flap raised by the patient while chopping wood. The photograph was taken twelve days after the injury and shows a swollen tense flap with necrosis of its edge. Figure 23 shows the healed state one month later; the flap is still tense and oedematous.

Figure 24 shows a large multiple palmar flap raised by trauma. The flap was sufficiently thick to expose the palmar muscles. The photograph was taken six days after the injury

FIG. 17

FIG. 18

FIG. 19

Pulp loss. Figure 17—Initial state. Figure 18—Palmar flap for two weeks. Figure 19—Result.

and seems to show good primary healing, but the tip of the flap is discoloured. Figure 25 shows the state one month later; the tips of the flap have sloughed away and the distal portions are swollen and oedematous. Figure 26 shows the final state fifteen weeks after the original injury.

The common factor in these two cases is that the flap is based distally. In the hand,

FIG. 20

FIG. 21

Pulp loss from middle finger; avulsion of distal phalanx of ring finger. Figure 20—Initial state. Figure 21—Result.

a flap which is based proximally has both an adequate arterial supply and a good venous return. A flap based distally, though it has an adequate arterial supply, has its normal venous return system completely severed. The flap bleeds freely from its tip but because of

FIGS. 22 and 23
Skin-flap injury before and after treatment.

FIG. 24 FIG. 25 FIG. 26
Skin-flap injury of palm. Figure 24—Six days after injury. Figure 25—Four weeks later. Figure 26—Fifteen weeks after injury.

the lack of venous return the blood stagnates, the flap swells and distal portions often slough. The narrower the base of the flap and the more acute the angle of its tip, the more the flap is likely to be lost.

Unfortunately, an accurate prognosis for a flap cannot be given unless it is of the narrow peninsular type, when it is almost certain to die. Some guidance can be obtained from the fact that a Z-plasty flap is usually designed with an angle of not less than 60 degrees. A flap that seems certain to die should be flayed and replaced as a full thickness skin graft. Probably the best treatment for the doubtful flaps is to excise the area of loss when it is defined and apply a split-skin graft to the defect if the area warrants it.

COMMENT

The cases described here have been chosen to illustrate a cross-section of the types of injury treated in this department as out-patients. Average results have been shown rather than the occasional good result obtained in the more difficult cases.

All the operations were done under local anaesthesia, most being under digital block, but three patients were operated on under brachial block anaesthesia and were admitted overnight as beds happened to be available. With the exception of the more extensive tendon injuries, all the patients were treated by the senior house surgeon to the department or by the surgical dressers under his direction.

A major problem has been the frequent change of house surgeons, necessitating the repeated training of new ones. By insisting on the strict immediate classification of all wounds and on their treatment by a standard plan, we have found that a satisfactorily high standard of treatment can be maintained. This training has been greatly helped by copying Clarkson's (1951) system of wall cards to illustrate principles of treatment.

Before reorganising the treatment of these minor hand injuries, we found that most of the unsatisfactory long-term results were in cases of nail injuries and pulp loss. This was because frequently the former had been left to heal by granulation and the latter had been grafted with thin split-skin grafts which would not withstand the trauma to which they were subjected.

By immediate grafting of all nail injuries we are now getting good results, and in the use of the palmar flap we have yet to have an ungrateful patient. The only disability which appears to result is a somewhat distinctive set of finger prints.

By the use of " Tubegauz " dressings, protected when necessary by leather finger stalls, most patients with lacerations and free grafts have been kept at work and the remainder have returned to useful employment earlier than we have previously managed.

Accurate figures are impossible to obtain, particularly as some patients, having been issued with their Final Certificate, have been known to destroy it and to persuade the private practitioner to continue the issue of Intermediate Certificates. A further factor which often hinders an early return to work is protracted litigation, cases sometimes being brought for what are truly trivial injuries.

My thanks are due to Sir Reginald Watson-Jones for his interest in this work and to the house surgeons to the Department who treated the cases. I would also like to thank Mr R. F. Ruddick for his willing co-operation in securing the photographic records.

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WEDGE ARTHRODESIS OF THE WRIST

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The method of arthrodesis of the wrist here described was originally evolved by Mr E. P. Brockman in a case of severe flexion deformity of the wrist due to a long-standing Volkmann's ischaemic contracture. Subsequently the method was extended in its application to other cases of flexion deformity of the wrist and later was also used for arthrodesis of the wrist in cases without deformity.

Spike arthrodesis, devised and described by Higgs (1931), has now long been used in the operative treatment of hammer toe deformity. The spike of the proximal phalanx well inserted into the socket of the distal phalanx provides relatively large cancellous bone areas in intimate contact and good internal fixation. It is thus comparable to an impacted fracture and sound early union results. The arthrodesed toe is also shortened. In the arthrodesis of the wrist to be described, the lower end of the radius is shaped into a wedge and is then inserted into the carpus which has been split open like an oyster (Figs. 1 and 2). This arthrodesis is thus an extension of the spike arthrodesis adapted and modified to deal with a more complex joint. In addition to providing the pre-requisites for sound bony union, this

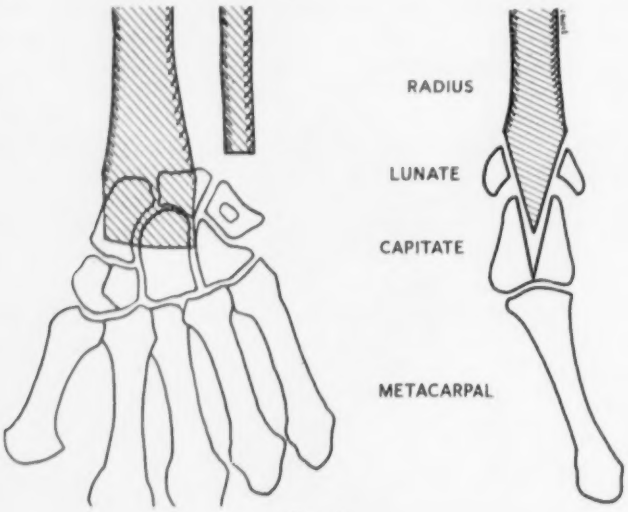

FIG. 1

Diagrams showing the position of the wedge in the split carpus.

arthrodesis similarly causes slight shortening of the forearm. The arthrodesis can be combined with resection of the lower end of the ulna when the return of rotation movements of the forearm is desired.

Of the many operations that have been described for obtaining wrist fusion, most have employed a dorsal approach and have included the use of a bone graft. The main variation in these techniques has been in the type of graft used. Thus onlay, inlay and sliding grafts, of compact or cancellous bone, from various sites and of varying shapes, have all been advocated (Abbott *et al.* 1942, Albee 1940, Brittain 1952, Colonna 1944, Butler 1949, Gill 1949,

Thomas 1950). Smith-Petersen (1940), extending the operation of Darrach (1936) for excision of the lower end of the ulna, described a lateral approach to the wrist, which was exposed through the bed of the resected ulna, the excised portion of bone being used as an inlay graft.

The multiplicity of these operations suggests that none has been found completely satisfactory. Pseudarthrosis and fracture are complications of the use of any graft, and in a recent review of wrist fusions by various methods Ross (1950) reported that fracture of the graft was not infrequent, particularly after Brittain's method of arthrodesis.

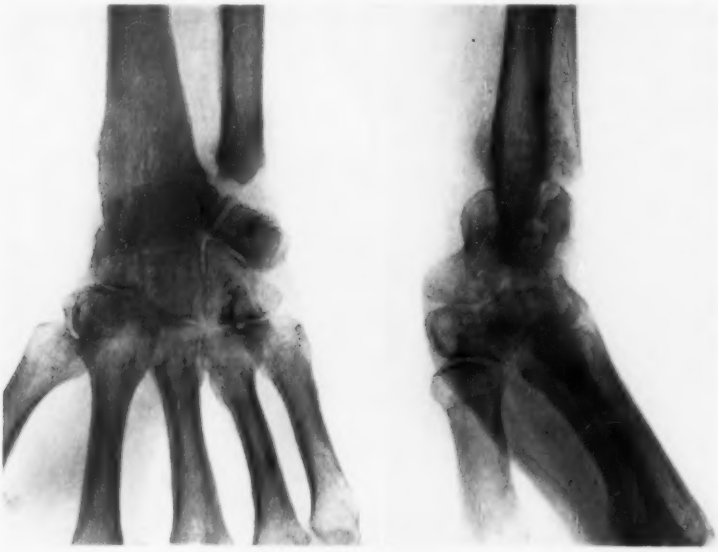

FIG. 2
Post-operative radiographs.

Wedge arthrodesis eliminates the need for a graft; and when combined with resection of the lower end of the ulna to preserve full rotation at the superior radio-ulnar joint, it has proved a most satisfactory operation for routine wrist fusion. It appears to compare favourably with all other methods. Nissen (1951) strongly advocated its use, and briefly described the operation with minor technical differences.

TECHNIQUE OF OPERATION

A dry operative field can be maintained by the use of a pneumatic cuff applied to the upper arm. But this increases post-operative swelling, which has occasionally led to displacement of the wedge. It is therefore preferable not to use a pneumatic cuff. The amount of bleeding is small and easily controlled, and it does not interfere with the technique of the operation.

Through a four-inch longitudinal incision in the mid-line over the dorsum of the wrist, the extensor tendons are identified and retracted. The capsule of the wrist joint is divided transversely. (It is imperative not to open the back of the intercarpal joints by mistake, for the proximal row of the carpus then becomes impossible to control later in the operation.) The lower ends of the radius and ulna are delivered into the wound and the soft tissues stripped from their volar surfaces (Fig. 3). The wrist is now further flexed until the palm of the hand lies parallel to the forearm. If it is intended to preserve or attempt to increase the range of rotation of the forearm, the lower inch of the ulna is excised. If no rotation is expected or desired, the ulna is included in the forearm wedge.

With an osteotome, the radius is shaped into a wedge of dense cancellous bone, tapered to as fine a cutting edge as possible. The carpus is then divided transversely with an osteotome so that the two halves can be opened up like an oyster (Fig. 4). The cleft should extend well into the second row of the carpus and in particular into the capitate. It is at this stage that the importance of preserving the capsule and ligaments of the carpus is realised, for if these have been inadvertently damaged, the carpus tends to disintegrate and a firm fit of the wedge cannot be obtained. The wedge is now inserted into the carpus, using the osteotome as a guide, and pushed home. It should fit firmly into the cleft (Fig. 5). The retracted extensor tendons are released, a few anchoring sutures approximate the extensor retinaculum and subcutaneous tissues, and the skin is closed. With the wrist held in a neutral position between flexion and extension, an above-elbow padded plaster is applied and split dorsally to allow for subsequent swelling.

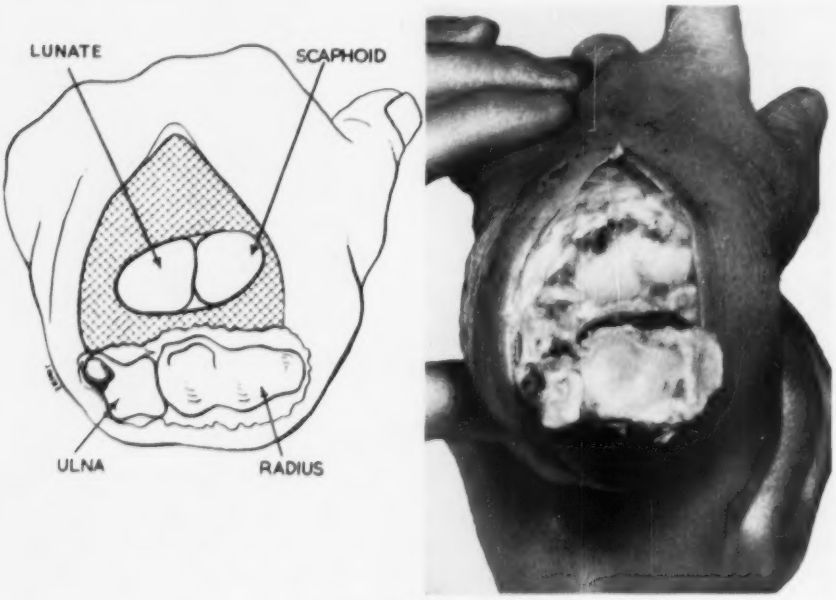

FIG. 3

The capsule of the radio-carpal joint has been divided transversely, and the wrist flexed through 180 degrees. Pronator quadratus can be seen stripped from the volar surface of the radius. In this case the ulnar styloid process has been avulsed by the medial ligament when the wrist was fully flexed.

Post-operative treatment—Elevation of the arm during the first few days assists in limiting the inevitable swelling of the hand and fingers. A radiograph is taken to confirm the position of the wedge in the carpus. Three weeks after operation the plaster is removed and the sutures are removed. A well moulded plaster is applied over stockinet and remains on for a total period of about three months, by which time union can be expected and further external support is seldom required.

CLINICAL MATERIAL

Eighteen patients in whom nineteen wrists had been arthrodesed by this method were available for review. Eight patients were male and ten were female. Their ages at operation varied from eight to fifty-five years. The length of follow-up varied from one to eighteen years.

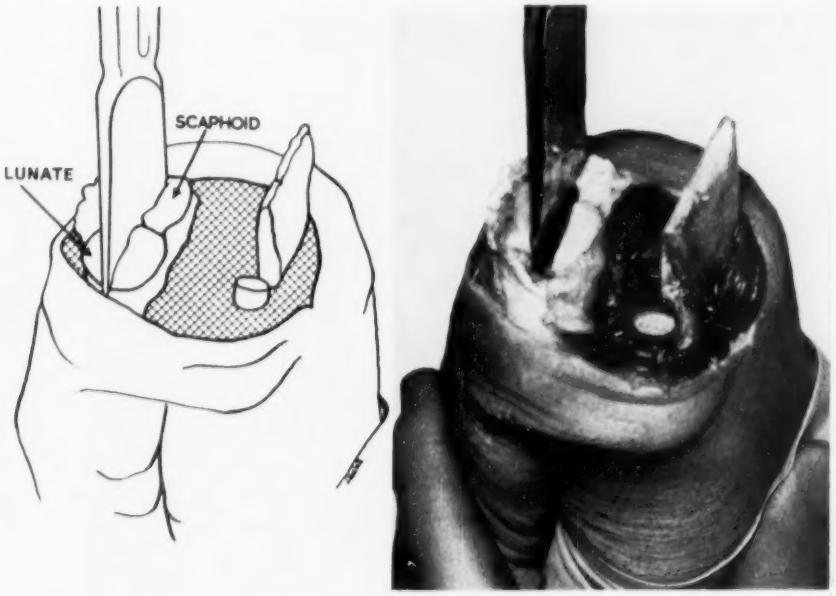

FIG. 4

The lower end of the radius has been tapered to a wedge and the distal inch of the ulna resected. The carpus is being split with an osteotome.

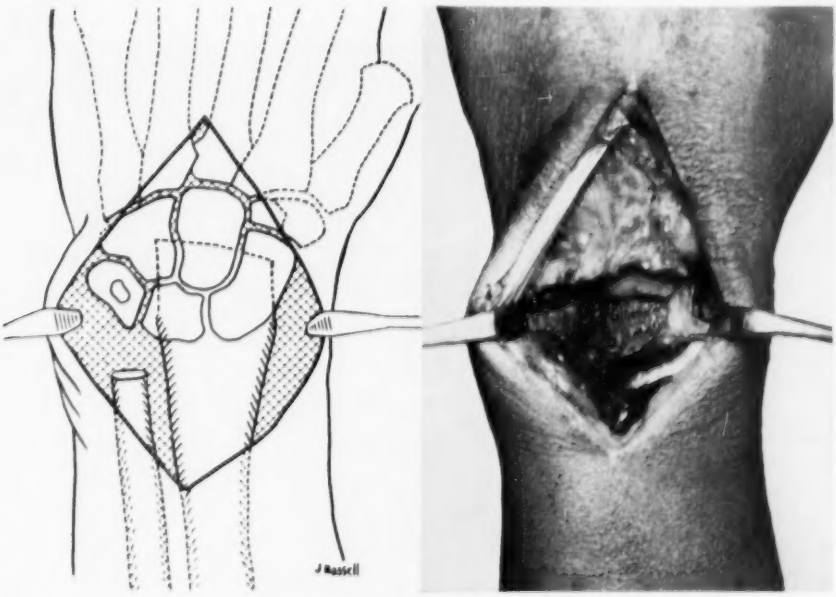

FIG. 5

The " wedge " has been inserted into the divided carpus, and fits securely. The resected lower end of ulna is seen.

The cases fall into two main groups. In the first group of twelve, flexion deformity of the wrist was the essential problem (Table I). The causes were Volkmann's ischaemic contracture (two cases), spastic hemiplegia (seven cases), brachial plexus birth injury (one case), and

TABLE I

ANALYSIS OF TWELVE CASES WITH FLEXION DEFORMITY OF THE WRIST

Case	Sex	Diagnosis	Age at operation	Length of follow-up (*years*)
1	F.	Volkmann's ischaemic contracture	14	18
2	M.	Volkmann's ischaemic contracture	12	17
3	F.	Brachial plexus birth injury	21 (re-operation 22)	6
4	F.	Arthrogryposis multiplex congenita	16	6
5	F.	Arthrogryposis multiplex congenita	Left 20 Right 20	5
6	F.	Spastic hemiplegia	19	10
7	F.	Spastic hemiplegia	12	6
8	M.	Spastic hemiplegia	19	4
9	M.	Spastic hemiplegia	26	4
10	M.	Spastic hemiplegia	17	4
11	M.	Spastic hemiplegia	8 (re-operation 15)	4
12	F.	Spastic hemiplegia	17	3

TABLE II

ANALYSIS OF SIX CASES WITHOUT FLEXION DEFORMITY OF THE WRIST

Case	Sex	Diagnosis	Age at operation	Length of follow-up (*years*)
Traumatic arthritis				
1	M.	Fracture of scaphoid	57	7
2	F.	Fracture lower end of radius	45	6
3	F.	Kienböck's disease	48	4
4	M.	Fracture of scaphoid	55	2
5	M.	Fracture of scaphoid	42	1
Chronic non-specific arthritis				
6	F.		55	3

arthrogryposis multiplex congenita (two cases—one bilateral). In the second group of six, pain with consequent loss of function was the essential problem (Table II). The causes were traumatic arthritis of the wrist (five cases) and chronic non-specific arthritis (one case).

FIG. 6

Case 9—Congenital spastic hemiplegia, showing the appearance and function of the hand before operation.

FIG. 7

Case 9—Condition after operation.

RESULTS

Cases with flexion deformity of the wrist—In all a sound fusion was obtained. The position of rotation of the forearm varied with the pre-existing deformity. Most wrists were arthrodesed in a pronated or semi-pronated position. The appearance of these hands was enormously improved and some improvement in function occurred in all (Figs. 6 and 7).

FIG. 8

Case 5—Arthrogryposis multiplex congenita, before operation.

FIG. 9

Case 5—Condition after operation.

In four cases resection of the lower end of the ulna had been performed, but in only one was rotation of the forearm increased.

In assessing the results of an operation of this kind the patient's views must be regarded as at least equal in importance to those of the surgeon. This is particularly true when the operation is undertaken primarily for the correction of an ugly deformity. All patients in this group were well pleased with the improvement in appearance of their hands, which was more important to them than the modest gain in function (Figs. 8 and 9).

It is perhaps worth recording that three women (one with Volkmann's ischaemic contracture, one with spastic hemiplegia, and one with arthrogryposis multiplex congenita) were married after their operations. All were able to run their own homes and look after their families. One other woman, with spastic hemiplegia, subsequently obtained a better job as a result of her operation. One young man with Volkmann's ischaemic contracture felt able for the first time to enjoy playing team games, another with spastic hemiplegia took up ballroom dancing as a pastime, and a third, also with spastic hemiplegia, became engaged while in his plaster splint, married soon after it was removed, and was able later to obtain a better job. These personal testimonies reflect, far better than any figures can, the difference in these patients' outlook on life when an unsightly deformity was rendered less conspicuous.

FIG. 10

Case 12—Radiograph nine years after operation. Fusion of radius with proximal row of carpal bones.

FIG. 11

Case 3—Radiograph four years after operation. Fusion of radius with both rows of carpal bones.

Cases without flexion deformity—Resection of the lower end of the ulna was performed in all these cases, and in all a sound arthrodesis was obtained with complete relief of pain. In five cases the functional result was excellent, full pronation and supination were preserved, and the patients were well satisfied with the result. In one case, though a sound fusion was gained, loss of function persisted; this patient suffered from post-encephalitic Parkinsonism, which probably accounted for the poor functional result.

Late radiographic appearances—In reviewing the late appearances of these arthrodeses, it was noted that in some cases fusion of the forearm wedge had occurred with only the proximal carpal row (Fig. 10), whereas in others there was sound fusion with both carpal rows (Fig. 11). Clinically these could not be distinguished.

Serial radiographs also showed well the remodelling that occurs at the site of arthrodesis. Though the original wedge of the radius remains visible for a year or longer, ultimately it becomes completely incorporated and indistinguishable in the new bone structure.

Complications—Displacement of the wedge occurred in two cases. In both the arthrodesis had been performed for flexion deformity of the wrist. In one, the immediate cause was excessive post-operative swelling, which necessitated wide splitting of the plaster. The other occurred for no certain cause in the youngest patient in this series—a boy of eight. In both it is probable that the wedge of the radius and ulna had not been inserted far or firmly enough into the distal carpal row. In the second case the patient was too young for this operation, for the small size of the bones made the operation technically more difficult. For this reason, and also because of the additional shortening of the forearm that will occur from premature fusion of the epiphyses, the operation should be deferred, when possible, until bone growth is almost complete. In both cases sound fusion was secured after a further similar operation.

Excessive mobility of the resected lower end of ulna was noted in one case. This caused only slight discomfort.

SUMMARY

1. A simple and effective method of wrist arthrodesis is described. Originally designed for the correction of flexion deformity of the wrist, it is useful also as a routine method of wrist fusion.

2. The results in nineteen cases are reviewed.

I wish to record my thanks to Mr E. P. Brockman for his help and encouragement in the preparation of this paper, and for permission to review his cases. My thanks are also due to the Photographic Department of Westminster Hospital for assistance with the illustrations.

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A SEROLOGICAL TEST IN THE DIAGNOSIS OF STAPHYLOCOCCAL INFECTION

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From the Royal National Orthopaedic Hospital and the Institute of Orthopaedics

The diagnosis of occult staphylococcal infection of bone is often difficult. Two cases are presented that illustrate the use of the anti-alpha haemolysin titration. In the second case this test appears to have been of value as a guide to effective chemotherapy. Such cases are now infrequent and we publish these results in the hope that further knowledge of the efficiency of this test may be sought elsewhere.

Fig. 1

Fig. 2

Case 1. Figure 1—Early radiograph showing destructive changes at the T.8-9 interspace and similar changes, less clearly shown, between T.7-8. Figure 2—Final radiograph, showing fusion of the affected vertebral bodies.

CASE REPORTS

Case 1—A man aged forty-seven was admitted to hospital in 1945 having been sent home from Italy with a diagnosis of typhoid fever and pleurisy. He had complained of backache, and radiographs showed a supposedly tuberculous lesion involving the seventh, eighth and ninth thoracic vertebrae (Fig. 1).

He was treated in a plaster bed for ten months, and since the disease then appeared quiescent he was discharged wearing a spinal support. Six months later he developed an occipital abscess, which was incised, and culture of the pus produced a profuse growth of staphylococcus aureus but no tubercle bacilli.

Two years later he was readmitted with pain and swelling in the left knee. This was caused by an abscess, and culture of the pus again grew staphylococci but no tubercle bacilli.

Four years after the onset of the spinal lesion he suffered girdle pain in the eighth and ninth

thoracic dermatomes. He was admitted to hospital. A gradually increasing paraplegia was discovered. Radiographs of the spine showed a sclerosing lesion of the sixth, seventh, eighth and ninth thoracic vertebrae. It was now thought that the whole thoracic disease might be staphylococcal osteomyelitis of the spine and that the paraplegia was secondary to this. The anti-alpha haemolysin titre was estimated for the first time and proved to be 25 units per millilitre (normal—less than 2 units per millilitre) and the leucocyte count rose from 15,000 to 17,000 cells per cubic millimetre (70 per cent polymorphonuclear leucocytes). Penicillin (100,000 units eight-hourly) was given for eighteen days, and the paraplegia rapidly receded. Power in the legs became normal and control of the sphincters returned. One month after the onset of paraplegia the anti-alpha haemolysin level had fallen to 20 units and a month later to 16 units (Fig. 3).

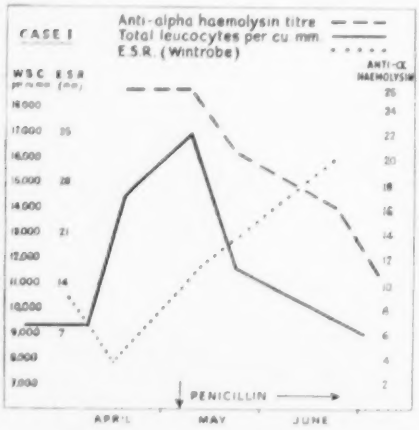

FIG. 3

Case 1.—Graphs showing alterations of anti-alpha haemolysin titre, leucocyte count and erythrocyte sedimentation rate.

The patient was discharged from hospital twenty-one weeks after the onset of the paraplegia and was walking with slight ataxia. He is now perfectly well and works as a plumber and decorator. Radiographs show healing of the diseased area with bony fusion (Fig. 2).

Case 2—A man aged fifty, who had had sciatica at intervals for seven years, was admitted to hospital in 1953 for exploration of the intervertebral discs. A large prolapse of the disc between the fourth and fifth lumbar vertebrae was removed completely. Operation was followed by fever and malaise, with a white cell count of 8,800 (72 per cent polymorphonuclear leucocytes). The temperature rapidly subsided with a short course of penicillin, but nine days after operation the wound was inflamed and there was a discharge of thin, serous fluid yielding a growth of staphylococcus pyogenes sensitive to aureomycin, chloromycetin and streptomycin. With a course of aureomycin, the inflammation subsided and the discharge ceased. The patient was discharged from hospital one month after the operation.

Three months after operation he developed a pyrexia of 103·6 degrees Fahrenheit, with tachycardia and a generalised aching pain in back, legs and left shoulder. There was no evidence of local infection at this time. The anti-alpha haemolysin titre was 2 units and the white cell count was 7,000 with 84 per cent polymorphonuclear leucocytes. Blood culture was sterile and the sedimentation rate was 7 millimetres/hour (Wintrobe). Radiographs of the lumbar spine showed no abnormality.

Three weeks after the onset of the pyrexia sciatica recurred, but there was still no sign of local infection in the wound. The anti-alpha haemolysin level had risen to 4–8 units per millilitre, and one week later was 16–32 units per millilitre (Fig. 4). Radiographs now showed erosion of the upper surface of the body of the fifth lumbar vertebra—an appearance suggestive of acute osteomyelitis (Fig. 5). The white cell count had risen to 10,750 cells per cubic millimetre and the sedimentation rate was 42 millimetres/hour. A course of aureomycin was given for three weeks and then changed to erythromycin. Further radiographs showed erosion of the lower surface of the body of the fourth lumbar vertebra with an abscess alongside the vertebral body (Fig. 6). But the patient felt less pain and his general condition had improved. The white cell count had fallen to 7,050 and the sedimentation rate was

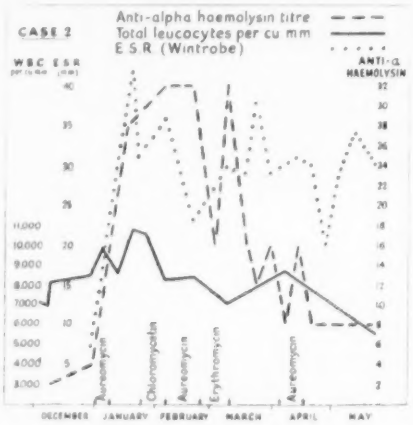

FIG. 4

Case 2.—Graphs showing alterations of anti-alpha haemolysin titre, leucocyte count and erythrocyte sedimentation rate.

30 millimetres/hour. Gradually the back pain and sciatica decreased and he was able to move about in a plastic jacket. Later radiographs showed satisfactory healing of the spinal lesion, and the anti-alpha haemolysin titre had fallen to 8–10 units per millilitre, the sedimentation rate to 29 millimetres/hour and the white cell count to 5,500 per cubic millimetre.

The patient is now free from pain and engaged in his normal employment as a greengrocer.

Comment—The first point of interest is that in spite of the absence of any clear physical signs or of a significant leucocytosis, the cause of this patient's symptoms was indicated by a steadily rising anti-alpha haemolysin titre (Fig. 4). This rose to a level, unprecedented in our experience, of 32 units in spite of a course of aureomycin and chloromycetin. It was decided that the maintenance of this high level of persistence of clinical symptoms indicated that the infection was still active; so the patient was given erythromycin. After this the

FIG. 5

FIG. 6

Case 2. Figure 5.—Radiograph four months after operation for removal of a prolapsed intervertebral disc. Note erosion of upper margin of L.5 vertebral body. Figure 6.—Four weeks later. Further erosion, now involving the lower margin of the body of L.4.

patient's general condition rapidly improved and his antibody level fell to 8 units, at which it has been maintained.

It will be noted on the chart that though the rise in sedimentation rate bears some relation to the rise of antibody, only a very mild rise was observed in the polymorph count and, had this alone been considered, the diagnosis would have been obscure. The second feature of interest is the fall in antibody titre after treatment by erythromycin when it had remained high after other antibiotics.

DISCUSSION

Serological tests in the diagnosis of staphylococcal infection have been of limited use. The reasons for this are too numerous to discuss in this communication and are dealt with in a publication in the press. The test which is most satisfactory from a laboratory point of view is probably the one used here, but all that can be said about it is that it seldom gives so

clear-cut an answer as in these cases. In Table I we set out the anti-alpha haemolysin titres of 259 hospital patients, and it will be seen that in more than half of those with staphylococcal osteomyelitis the titre was not raised. In our experience about 20 per cent of the staphylococci examined do not produce alpha haemolysin; so the infection they cause would not be accompanied by a rise in the specific antibody. On the other hand, it is rare to find a patient with a titre of over 8 units who has not recently had a severe staphylococcal infection.

TABLE I

ANTI-ALPHA HAEMOLYSIN TITRES OF 259 HOSPITAL PATIENTS

	Osteomyelitis	Secondary staphylococcal infection	No staphylococcal infection
Less than 2 units per millilitre	30	20	140
2–4 units per millilitre	9	6	23
4–8 units per millilitre	5	7	5
8–16 units per millilitre	6	4	1
16–32 units per millilitre	1	1	1

Dolman (1935) reported that the normal value for healthy individuals is from 0·3–1 international unit and Murray's (1935) figures were 0·4–2 international units. Both obtained universally high titres in osteomyelitis. Blair and Hallman (1935) in their study of staphylococcal haemolysin in osteomyelitis and other staphylococcal infections found that two-thirds of the sera from the cases of osteomyelitis had a titre of one international unit or less, and only one-fifth of the sera had definitely higher titre averaging 5·1 units, the highest being 17·7 units. They concluded that a high anti-alpha haemolysin titre was not characteristic of staphylococcal osteomyelitis.

We believe that this test is worth doing when the staphylococcal etiology of a bone and joint lesion is under consideration and we would give considerable weight to a rising titre or a titre of over 10 units as favouring a staphylococcal origin.

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OSTEOCHONDRITIS DISSECANS IN THREE MEMBERS OF ONE FAMILY

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Osteochondritis dissecans affecting a single joint is relatively common. Multiple lesions are less common and the occurrence of osteochondritis dissecans in several members of a family is sufficiently rare to warrant description.

Wagoner and Cohn (1931) described osteochondritis dissecans of the knee in the son, father and paternal uncle of one family and in both knees of two brothers. Bernstein (1925) recorded bilateral affection of the knee in two sisters and a brother, and Novotny (1952) reported osteochondritis of one knee in each of two brothers.

FIG. 1

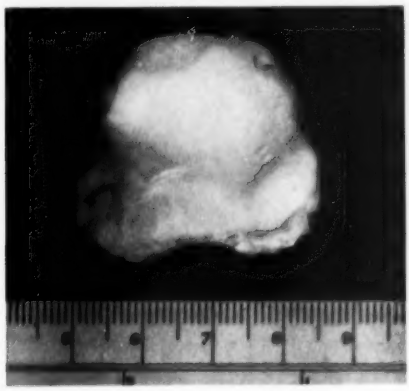

FIG. 2

Case 1. Figure 1—Areas of osteochondritis dissecans in medial femoral condyle. Prominent tibial spines and early osteoarthritic lipping. Figure 2—Fragment of articular cartilage removed from left knee.

The following report refers to a family of three boys and a girl, of whom two of the brothers and the sister were affected.

Case 1—This first patient, a boy aged nineteen years when first seen, complained of a limp, pain, and swelling of the left knee of several years' duration, particularly troublesome during the last six months. There was no history of injury. On examination, an effusion was present in the left knee and there was a block to full flexion and to full extension of the joint. *Radiographs* showed osteochondritis dissecans of the medial femoral condyles of both knees, prominent tibial spines and early osteoarthritic lipping (Fig. 1). The radiographs of other joints were normal. The left knee was explored and a large defect was found on the weight-bearing surface of the medial femoral condyle. A fragment of cartilage measuring 3·6 × 2·4 centimetres and comprising most of the articular surface of the medial condyle of the femur was almost completely separated from the underlying bone (Fig. 2). The fragment was

FIG. 3

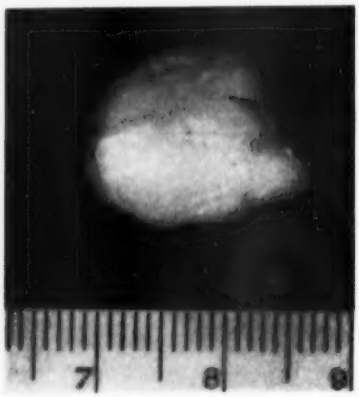

FIG. 4

Case 2. Figure 3.—Area of osteochondritis dissecans in medial femoral condyle. Figure 4.—Fragment of articular cartilage removed from left knee.

FIG. 5

FIG. 6

Case 3. Figure 5.—Areas of osteochondritis dissecans in right medial femoral condyle. Figure 6.—Similar lesion in left medial femoral condyle.

removed, leaving a deep crater, the edges of which were smoothed off. No other lesion was noted. When last seen a year and a half later he complained of occasional pain in both knees. In neither joint was there an effusion nor a block to full extension. The right knee could be flexed to 65 degrees and the left to 70 degrees.

Case 2—The girl, two years younger than the first patient, was also aged nineteen years when she was first seen. She gave a history of recurrent pain in the left knee accompanied by occasional locking of the joint of five years' duration. Symptoms began when the knee locked while she was doing a high jump. *Radiographs* taken in another hospital showed osteochondritis dissecans in the left medial femoral condyle. Operation was not advised. At the time of first examination there were no physical signs apart from a soft click on extending the knee from a fully flexed position. Radiographs (Fig. 3) confirmed the presence of osteochondritis dissecans. Other joints were normal. The left knee was explored, and an oval area of osteochondritis dissecans measuring $1{\cdot}6 \times 1{\cdot}2$ centimetres was seen on the lateral aspect of the medial femoral condyle (Fig. 4). The cartilage was almost free of the underlying bone, a few filmy bands of soft tissue holding it in its bed, from which it could easily be tilted. The fragment was removed. On the weight-bearing surface of the lateral femoral condyle there was a flattened area of articular cartilage measuring $2{\cdot}2 \times 1{\cdot}7$ centimetres which had lost its normal sheen and was clearly demarcated from the surrounding cartilage; although there was no actual separation from the underlying bone or indentation on pressure it seemed likely that the fragment would ultimately become detached. No other abnormality was noted. When she was last seen a year and a half after operation there was a full range of painless movement in the knee joint. There was no radiographic evidence of separation of the diseased cartilage in the lateral condyle.

Case 3—This patient, a younger brother of the other two, was aged eighteen years when first seen. He gave a history of occasional discomfort in the left knee over several years. Clinical examination was negative but radiographs showed bilateral osteochondritis dissecans of the medial femoral condyles (Figs. 5 and 6). No abnormality was noted in any other joint.

Examination of other relatives—The third brother was a mental defective but neither he nor the father and mother who were examined and radiographed showed any evidence of osteochondritis or loose body in any joint. So far as was known the paternal and maternal grandparents had no joint trouble. There was no blood relationship between the parents.

Comment—The etiology of osteochondritis is unknown. It seems, however, from the cases described and from those of Bernstein, of Novotny, and of Wagoner and Cohn that there is in some cases a familial factor.

SUMMARY

Three cases of osteochondritis dissecans of the knee in two brothers and a sister are described. In the brothers both knees were involved. None of the other joints of the skeleton was affected in any of the patients.

I wish to express my thanks to Mr Roland Barnes for his help and criticism.

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FAMILIAL OSTEOCHONDRITIS DISSECANS

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The etiology of osteochondritis dissecans is unknown. Two hypotheses have been advanced—the traumatic and the embolic.

In 1933 Fairbank reviewed the literature and came to the conclusion that trauma was the most likely underlying cause. Smillie (1951) supported the traumatic theory and was impressed by the role played by a prominent tibial spine in osteochondritis dissecans of the knee. In 1912 Axhausen first proposed that the typical lesion could be embolic in origin. Such a hypothesis was advanced as a reasonable explanation of some cases of osteochondritis dissecans by Watson-Jones (1952), who suggested that the emboli might consist of rouleaux of red corpuscles that are formed whenever there is a rise in the blood sedimentation rate.

In 1925 Bernstein described osteochondritis dissecans of the knees in two sisters and a brother and in 1931 Wagoner and Cohn recorded bilateral osteochondritis dissecans of the knee in a boy, his father and his uncle. In each of the papers the suggestion of an underlying hereditary or constitutional factor was made.

The cases described here are those of a mother and three of her four daughters, all of whom suffer from the disease.

CASE REPORTS

In 1951 the second daughter, Doris, aged thirteen, was brought to hospital with a locked right elbow. Radiographs showed the typical appearance of bilateral osteochondritis dissecans of the capitulum with a loose body on the right side (Fig. 1). Radiographs of both hips, knees and ankles revealed no abnormalities. The offending loose body was removed and there have been no further locking incidents, although a recent radiograph reveals a second loose body within the joint.

A week later the eldest sister, Betty, aged fourteen, was seen with a swollen left knee. Investigation showed that she was suffering from osteochondritis dissecans of both knees (Fig. 2). The elbows were radiologically normal. After locking of first one and then the second knee, a loose body was removed from each. She has remained free from symptoms.

The two youngest sisters, Valerie and Vera, were examined. Valerie, aged ten, had no symptoms in any joint. Radiographs, however, revealed the typical changes of osteochondritis dissecans of both medial femoral condyles (Fig. 3). The elbows were radiologically normal. In the two years since her first examination she has remained free from symptoms.

Vera, aged three, was found to have no clinical or radiological evidence of osteochondritis dissecans in knees or elbows. Nor was there evidence of the disease on re-examination two years later.

The mother of the four girls, aged forty, remembered having had recurrent swelling of the knees during her 'teens, and said that both knees used to let her down. In recent years she has suffered from "rheumatism." Clinically there was osteoarthritis of both knees, and radiographs showed bilateral diminution of the joint spaces, with evidence of old erosion of the femoral condyles and the presence of small loose bodies (Fig. 4). These findings suggest that in her youth she also suffered from osteochondritis dissecans.

The mother is only four feet six inches in height, and all the children are about six inches below the normal height for age. In other respects they are normally developed.

DISCUSSION

The occurrence of osteochondritis dissecans in more than one member of a family is unusual. The fact that it does sometimes occur lends weight to the suggestion that in many

FIG. 1

Doris. Aged twelve. Osteochondritis dissecans of the capitulum in both elbows.

FIG. 2

Betty. Aged fourteen. Osteochondritis dissecans of the medial femoral condyles of both knee joints.

of these cases, especially when more than one joint is involved, there is an underlying constitutional or developmental factor.

The case of Valerie, the girl aged ten, is of particular interest in that the radiographic

changes of osteochondritis dissecans have been present for three years, but she has remained symptomless without treatment. This suggests that the disease may begin at an early age, remaining silent until late childhood, or that it may never gain a clinical identity.

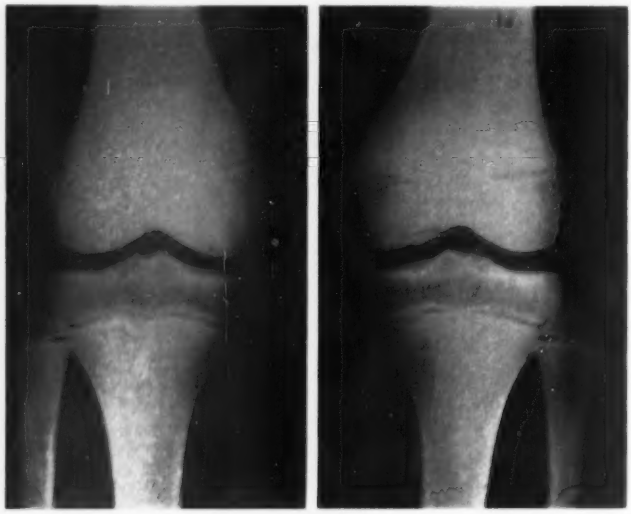

FIG. 3

Valerie. Aged ten. Typical radiological changes of osteochondritis dissecans in both knee joints.

FIG. 4

The mother of the girls. Aged forty. Erosion of both medial femoral condyles, with small loose bodies and secondary degenerative changes.

The association of dwarfism with osteochondritis dissecans in one case (Mr Roaf's) was recorded by Roberts and Hughes (1950) in their "Clinical Study of Osteochondritis Dissecans

of the Elbow Joint." In that patient, a girl of eleven, both elbows, both patellae, and both medial femoral condyles were affected.

SUMMARY

Osteochondritis dissecans occurring bilaterally in either the knees or the elbows is recorded in four members of one family. The suggestion is supported that the underlying pathology may be developmental, resulting in a form of localised osteochondrodystrophy.

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AN UNUSUAL FRACTURE-DISLOCATION OF THE CARPUS

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A man aged thirty-one fell ten feet and on breaking his fall injured his right wrist. Radiographs the day after the injury showed a fracture of the scaphoid in good position and a fracture of the capitate with the proximal fragment inverted (Fig. 1). No reduction

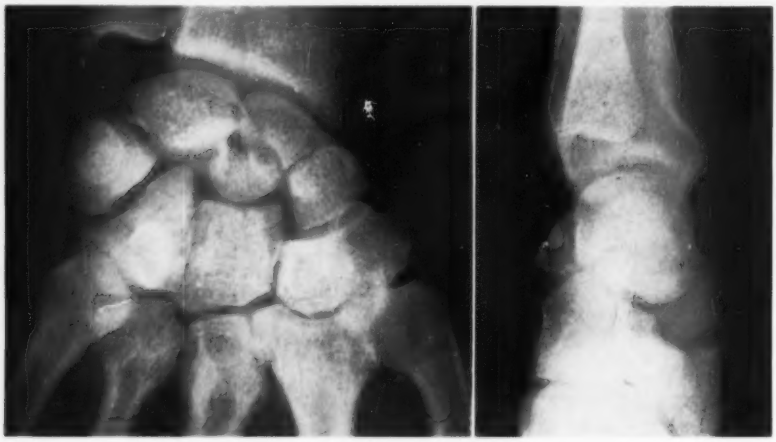

Fig. 1
Initial radiograph.

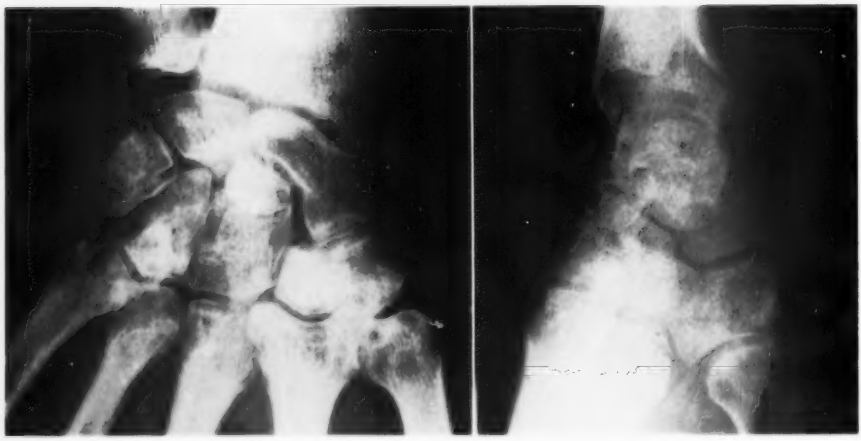

Fig. 2
Four months after injury.

had been carried out, but the injury is thought to have been a trans-scaphoid, trans-capitate perilunar fracture-dislocation of the carpus which underwent spontaneous reduction with inversion of the proximal fragment of the capitate. Owing to the instability of the carpus

and the importance of getting union of the scaphoid, it was decided to leave the capitate alone until the scaphoid was united. Immobilisation in plaster was continued for four months, by which time radiographs showed early union of the scaphoid, but with evidence of avascular changes in the proximal pole of the capitate (Fig. 2).

FIG. 3
One year after injury.

Further treatment was by active use and exercises. A year after the injury he was using the hand normally without pain on heavy use. There was about three-quarters of the normal range of movement at the wrist. He had a powerful grip and had no discomfort when he took pressure on the hand in dorsiflexion. Radiographs at that time showed that the proximal fragment of the capitate had united in the inverted position and had regained its normal density (Fig. 3). The scaphoid was sound and the calcification of the carpus was good.

I am indebted to Mr M. Morel, who treated this patient in consultation with me.

GAMEKEEPER'S THUMB

C. S. Campbell, Bridge of Earn, Scotland

The articulation between the metacarpal and the proximal phalanx of the thumb is a hinge-like joint which depends for stability on the capsule reinforced by the extensor expansion, palmar accessory and collateral ligaments. It is the collateral ligaments which determine lateral stability in extension, and ligamentous insufficiency on the ulnar side as a result of injury causes impairment of the pincer action between thumb and forefinger (Watson-Jones 1943). It is the purpose of this article to show that a similar ligamentous insufficiency may exist in the absence of trauma in gamekeepers engaged in the killing of rabbits; and that the condition is so common amongst gamekeepers as to constitute an occupational characteristic.

Mechanism—The gamekeeper's method of killing a wounded rabbit is to hold the head in one hand and the buttocks (or rear legs) in the other. A strong pull is then exerted while the neck is sharply extended in the thumb-forefinger cleft. The strain of this manoeuvre should be taken in the cleft; but almost invariably a loose grip causes the neck to be stretched

Fig. 1

Fig. 2

A gamekeeper's method of killing a rabbit by stretching and hyperextending the neck. The photograph on the right shows the method of gripping the neck in the thumb-forefinger cleft.

against the ulnar side of the thumb. It is the force of this pull, repeated manyfold, which stretches the collateral ligament (Figs. 1 and 2).

In the twenty-four gamekeepers examined, only four failed to show laxity of the metacarpo-phalangeal ligament on the ulnar side. The hand which held the rabbit's head was invariably the one affected. In this respect fifteen right and three left thumbs showed the deformity. The remaining two men, with lesions of both thumbs, were found to be ambidexterous. Of the four gamekeepers unaffected, one did no rabbit killing, one used a

different method and a third showed osteoarthritis of the first metacarpo-phalangeal joint in both hands. The fourth held the head of the rabbit well into the thumb-forefinger cleft. He alone used the manoeuvre described in the approved manner.

Pathology—The pathological anatomy of the condition resembles traumatic rupture of the collateral ligament when healing occurs with lengthening. Two forms are encountered. In the first, the ligament is lengthened by repeated stretching which allows the phalanx to tilt on the metacarpal when radial stress is applied (Fig. 3). The second type occurs as a

FIG. 3

FIG. 4

Figure 3—Tilting of the proximal phalanx on the metacarpal head permitted by laxity of the medial ligament. Figure 4—Continued pressure causes the tilted phalanx to slide laterally.

sequel to the first when continued pressure, brought to bear on the already lengthened ligament, causes the phalanx to slide over the metacarpal head. Both tilt and slide are thus present on application of the causative force (Fig. 4).

Clinical features—The severity of individual lesions was found to be directly related to the amount of rabbit killing undertaken by the gamekeeper. Symptoms, however, were not related to clinical signs. In this series a third of the patients complained of weakness of the thumb-forefinger grip and an ache in the metacarpo-phalangeal joint. The remaining two-thirds were unaware of an abnormality.

It is evident that there are other occupations in which a similar strain constantly applied to the inner side of the thumb could cause a comparable ligamentous laxity. For example, anaesthetists who maintain the face mask in position by hand do so with the collateral ligament on the ulnar side on stretch. A degree of ligamentous laxity has been noted in such circumstances. In general, however, the condition is so characteristic that a recent patient who denied being a gamekeeper later admitted an extensive interest in the poaching of rabbits.

I wish to thank Mr I. S. Smillie for his helpful criticism.

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THE FATE OF RADIOPAQUE MEDIA INJECTED INTO THE CANCELLOUS BONE OF THE EXTREMITIES

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From the Department of Anatomy, University of Liverpool

Since the work of Erhardt and Kneip (1943), and of Drasnar (1946), it has been known that solutions injected into cancellous bone are rapidly removed into the venous system. This has since been confirmed by Mallet-Guy and Micek (1949), Leger and Frileux (1950) and Olivier (1950) by injections of radiopaque media into the tibia and pubis. More recently Fischgold *et al.* (1952) and Tori (1954) found that the intravertebral and extravertebral plexuses of veins, and even the ascending lumbar vein and azygos system of veins, could be filled by 50 per cent or 70 per cent diodone very soon after its injection into a vertebral spinous process or a rib. Apart from this work by Continental investigators it does not appear that any observations have been made on the fate of materials injected directly into cancellous bone, except for those of Heald (1951) and others in an investigation of the method of treatment of fibrositis by Laughton-Scott. In view of the interesting radiographic appearances obtained by Heald (1951), it was decided to obtain further data on the cadaver after the injection of radiopaque media directly into cancellous bone.

Though the observations are as yet few, a preliminary communication seemed desirable in order to stimulate further investigations of this technique in the living subject.

MATERIAL AND METHODS

Observations were made on the cadavers of three men and three women, aged forty-six, fifty-three, sixty-one, sixty-three, sixty-eight and seventy-six years, death being due to coronary thrombosis in three cases, bronchopneumonia in two cases, and carcinoma of the bronchus in one. The experiments were performed forty-eight to seventy-two hours after death. In the first experiments Lipiodol was used as the injection medium, but it was later found that 50 per cent diodone (Pyelosil, Glaxo Laboratories Ltd.) could be injected more easily, because of its lower viscosity. It was finally discovered that a suspension of barium sulphate (Micropaque, Damancy & Co. Ltd.) could be used for injection and provided better contrast radiographically. The pressure necessary for injection was, however, slightly greater than with diodone.

The method of injection employed is like that of previous workers, and requires a sternal puncture or lumbar puncture needle ground so as to produce a symmetrical sharp point. This is pushed through the skin and tapped into the underlying bone with a hammer. The trocar can be withdrawn quite easily once it has perforated the compact bone and the needle is imbedded within the cancellous bone; till then, the trocar cannot be withdrawn except forcibly. In the living subject infiltration of the overlying skin with procaine makes the procedure quite painless (Plewes 1953). A syringe charged with radiopaque medium is attached to the needle, and the injection can then be made with ease.

Injections were made in a variety of situations, using 5–10 millilitres of radiopaque material. Much more could have been injected. The site of investigation was radiographed immediately after the injection, which never took more than forty-five seconds.

RESULTS

In several circumstances dissections were performed immediately after injection and radiography in order to determine exactly the route of drainage. In one experiment the skin

was reflected over the calcaneum, and injection made directly into the bone from the medial aspect; the opaque medium could then be seen passing rapidly into numerous minute veins, and from these directly into the dorsal venous arch of the foot, the long saphenous and posterior tibial veins. In a further experiment, during injection of 6 millilitres of Micropaque into the medial malleolus, the long saphenous vein could be seen to distend during the injection, and dissection of the leg afterwards showed that the suspension had travelled along the long saphenous vein as far as a point 17 centimetres proximal to the adductor tubercle of the femur. Further experiments, confirmed by dissection, showed that in every case in which the contrast medium was injected into cancellous bone, it was rapidly removed by small venous channels leaving the bone at various points near the site of injection.

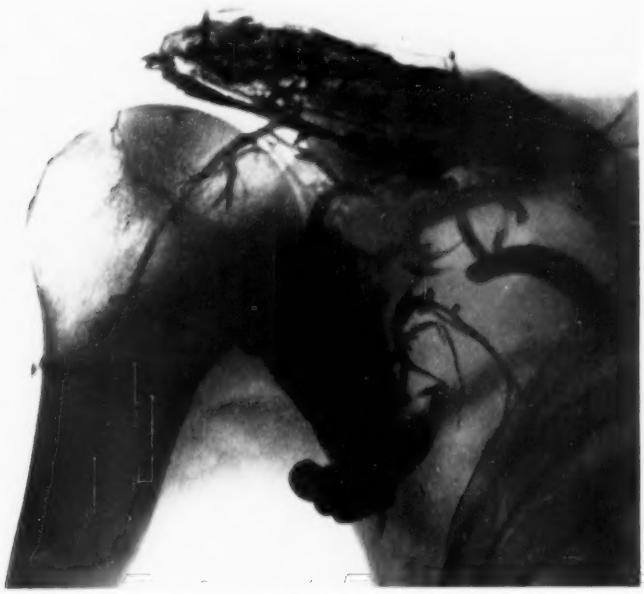

FIG. 1

Right shoulder region after injection of 5 millilitres Micropaque into the acromion process. The injection needle can be seen just medial to the tip of the acromion process, the intra-osseous channels in which are clearly shown. Only a short segment of the axillary vein, seen superimposed on the lateral border of the scapula, is filled; it is markedly distended and shows the outline of valves. A communicating vein between the veins of the acromion process and circumflex humeral veins is superimposed on the head of the humerus.

Several attempts were made to inject radiopaque media into the compact bone of the diaphysis in certain situations, such as the shaft of the tibia and of the humerus, but it was found impossible to penetrate the bone with the needle.

The upper extremity. *The acromion process of the scapula*—Five millilitres of Micropaque injected into the lateral extremity of the acromion process of the right scapula produced rapid filling of neighbouring veins and those in the axilla. The acromion process was drained by small and tortuous intra-osseous channels which collect into two larger extra-osseous veins; these drain into the cephalic and the axillary venous system (Fig. 1). The lateral channel draining the acromion process also filled the circumflex humeral veins in a retrograde manner, and the medial channel could be recognised as an acromial tributary of the cephalic vein. The contrast medium was seen in only a short segment of the axillary vein because the valves

prevented retrograde flow; in the lower part of this segment the valves could be clearly seen ballooning out at the lower part of the lateral border of the right scapula.

The head of the humerus—Ten millilitres of Micropaque were injected into the region of the lesser tuberosity of the left humerus. At the point of the needle the contrast medium filled the interstices of the cancellous bone to give a diffuse stellate appearance (Fig. 2). This region was chiefly drained by one large venous channel emptying into the cephalic vein. Here the distribution of Micropaque passed mainly proximally into the axillary vein, where the progress of the medium in a distal direction was again prevented by a valve at a similar site to that shown in Figure 1. The circumflex humeral vessel, outlined lateral to the axillary pool of contrast medium, presented one valve obstructing retrograde flow. It was observed

FIG. 2

Left shoulder region after injection of 10 millilitres Micropaque into the lesser tuberosity. Intra-osseous channels may be seen to be filled at the point of the needle, draining inferiorly into the cephalic vein (C), the radiopaque medium in which was found, by dissection, to be continuous with that in the axillary vein (A). The site of injection is also drained by a circumflex humeral vein (C.H.) which, in turn, drains into the axillary vein just proximal to the site of its distension caused by valves.

that vessels following this route of drainage are smaller than those reaching the cephalic vein. This would confirm the observations of Mallet-Guy and Micek (1949) that, in certain circumstances, superficial veins are demonstrated more readily than the deep veins by this method of phlebography.

The radiographic appearances found in this experiment were confirmed by subsequent dissection.

The olecranon process of the ulna—On injection of 3 millilitres of 50 per cent diodone into the olecranon process of the right ulna, the radiopaque medium was found to drain through various intra-osseous channels, and then into the cubital veins just below the site of injection (Fig. 3). The diodone also filled the cephalic and basilic veins some distance proximal to the elbow joint. The vein mainly involved in draining the upper end of the ulna is one which

leaves the bone just below the coronoid process, removed from the nutrient foramen of the ulna.

The lower end of the radius—Six millilitres of Micropaque introduced into the lower end of the right radius drained into venous channels as high as the elbow. The radiopaque medium filled the interosseous veins as well as the lower end of the cephalic vein (Fig. 4).

The lower extremity. *The ilium and greater trochanter*—In one experiment 6 millilitres of Micropaque were injected into the right greater trochanter; the radiopaque medium filled intra-osseous channels and then immediately drained into the circumflex femoral vessels and their branches, and eventually the femoral vein itself. Twenty minutes later a further

FIG. 3

FIG. 4

Figure 3—Elbow after injection of 3 millilitres diodone. The injection needle is seen penetrating the olecranon process. Intra-osseous channels draining inferiorly from the site of injection collect to form tributaries of the cubital veins which, in turn, drain into the cephalic and basilic veins. Dilations along the course of the veins indicate the presence of valves. Figure 4—Forearm after injection of 6 millilitres Micropaque into the lower end of the radius. Intra-osseous veins in the radius, the cephalic vein proximal and distal to the site of injection, and the interosseous veins, are well shown.

6 millilitres of Micropaque were injected into the right anterior superior iliac spine; branches of the superior gluteal and deep circumflex iliac veins were clearly shown immediately after injection. The composite radiograph (Fig. 5) demonstrated clearly the venous network in the right hip region. In this case, the deep veins draining this region have been injected.

Proximal end of the tibia—Four millilitres of diodone were injected into the proximal end of the right tibia just distal to the epiphysial line (Fig. 6). The popliteal vein was immediately and clearly outlined, together with the medial and lateral inferior genicular veins which appear to be draining the injection site. The short saphenous vein is also filled for some 12 centimetres distal to its termination.

The medial malleolus—Six millilitres of Micropaque were injected into the medial malleolus of the left tibia. Antero-posterior (Fig. 7) and lateral (Fig. 8) radiographs were taken within a minute. Both views show the delicate intra-osseous channels draining into small veins which on the medial side reach the long saphenous and posterior tibial veins, and on the lateral side the short saphenous vein. Subsequent dissection showed that the dorsal venous arch

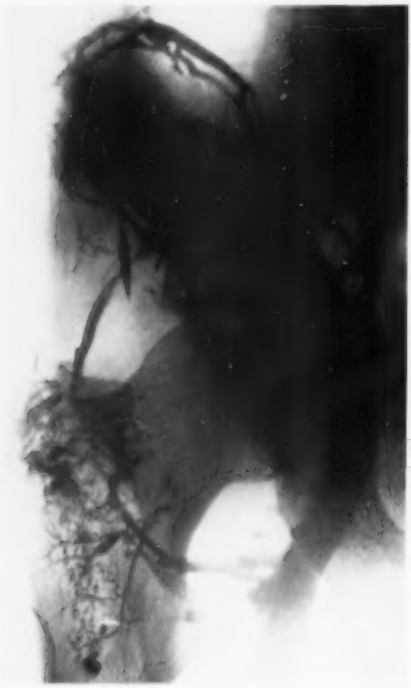

FIG. 5

FIG. 6

Figure 5—Hip region after injection of 6 millilitres Micropaque into the greater trochanter and 6 millilitres into the anterior superior iliac spine. Intra-osseous channels in the greater trochanter are particularly well shown, draining into the circumflex femoral vessels. Branches of the superior gluteal and deep circumflex iliac veins are also shown in relation to the iliac crest. The two sites of injection eventually drain into the femoral and external iliac veins.
Figure 6—Knee after injection of 4 millilitres diodone into the proximal end of the tibia just distal to the epiphysial line. The needle is seen end-on close to the tibial tubercle. The inferior genicular veins, short saphenous and popliteal veins are clearly shown.

of the foot was also filled with radiopaque medium. Reflux flow distally outlined the continuation of the posterior tibial veins on the medial aspect of the calcaneum. Proximally the vessels were more clearly outlined, and the valves in the venae comites of the posterior tibial artery were particularly well shown.

DISCUSSION

These researches confirm those of previous workers in demonstrating the rapidity of drainage into the venous system of solutions and suspensions introduced into cancellous bone. In the living subject, diodone introduced into the greater trochanter passes into the regional veins within half a minute of the termination of the injection, and is completely removed from these veins within three to four minutes after injection (Ross 1954). This may be an underestimate, however, since it was possible to introduce one and a quarter pints

of plasma through a sternal puncture needle into the manubrium sterni (which is composed of cancellous bone) of a collapsed woman in less than two minutes (Bailey 1947). Indeed, the sternum in the adult has been used as a site for blood transfusion (Tocantins and O'Neill 1941, Tocantins, O'Neill and Jones 1941), when the rate of flow has been up to 25 millilitres a minute with a head of pressure of 100–150 centimetres. This method of intrasternal transfusion was found useful during the last war because of the firm anchorage given by the sternum for the injection needle, the great vascularity of the sternum and its efficient drainage through internal mammary and innominate veins into the superior vena cava. The curious feature of the vascularisation of bone, however, is that, although the venous drainage is

FIG. 7

FIG. 8

Figure 7—Ankle region after injection of 6 millilitres Micropaque into the medial malleolus. The valves in the posterior tibial veins are well shown. Figure 8—Lateral view of the specimen shown in Figure 7. Reflux flow has outlined the continuation of the posterior tibial veins on the medial aspect of the calcaneum.

rapid, and quicker than that of soft tissues, the arterial supply through nutrient arteries is slow and at a low pressure (Lamas, Amado and Da Costa 1946).

The method of introduction of solutions and suspensions into cancellous bone is also of interest for the technique of phlebography. Apart from the investigations of Continental workers, this procedure does not appear to be generally recognised, although it is easily effected. The experiments here reported suggest that the venous system of a limb could be filled easily and safely through a needle inserted into cancellous bone at numerous sites. The simplicity and safety of this procedure (Tori 1954), and the fact that it can be used in oedematous limbs in which the superficial veins are not visible (Leger and Frileux 1950) further justify the clinical application of this technique. The danger of osteomyelitis after transfusion into the medullary cavity of infants (Gunz and Dean 1945, Ellison 1945) must, however, be remembered.

SUMMARY

Radiopaque solutions and suspensions introduced into cancellous bone in the extremities of the cadaver are rapidly removed into the venous system through regional superficial or deep veins. The experiments described in this communication confirm the simplicity of methods of introduction of fluids into cancellous bone and justify further observations on their clinical application for the technique of phlebography.

This research has been aided by a grant from the Medical Research Council. We wish to thank Mr C. FitzSimon for his technical assistance; and Glaxo Laboratories Ltd., and Damancy & Co. Ltd., for supplies of Pyelosil and Micropaque.

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FREDERIC WOOD JONES

1879-1954

When towards the end of last year Frederic Wood Jones died, we lost one of the most widely travelled, most original and most inspiring of philosophical anatomists, whose contributions to his subject and through it to many branches of medicine, including orthopaedics, have been far reaching. For some years before his death he was Conservator of the Hunterian Museum of our College, where he had followed Sir Arthur Keith and, like him, was a high court of appeal to whom anyone with a problem in anatomy, and particularly comparative anatomy, could turn and would not turn in vain. He was as great a naturalist as Richard Owen or Leidy were in their day and, like them, could reconstruct the whole animal from a few fossil remains, or from an odd bone dug up in one's garden.

When he came to the College as the first William Collins Professor of Human and Comparative Anatomy he had occupied chairs of anatomy in four universities, two in this country and two in Australia, and the Chair of Physical Anthropology in the University of Hawaii. There, with his colleague, Stanley Porteous, the Professor of Clinical Psychology,

he produced *The Matrix of the Mind*, a book in which the two authors attempted, with considerable success, to blend the two sciences of neurology and psychology by a mutual consideration of structure and function.

At the close of the first world war, in a series of lectures given as part of a course of instruction to officers of the Royal Army Medical Corps at the special Military Orthopaedic Hospital at Hammersmith (now the Postgraduate Hospital) he dealt with the principles of anatomy as seen in the hand. These lectures were subsequently published as a book, and some years later he produced a corresponding volume on the foot. No orthopaedic surgeon with a real interest in his subject can afford not to study these two books in which consideration is given to such subjects as joints and the actions of muscles as well as to the detailed anatomy of the extremities.

Wood Jones upset some of the predominant views on evolution held by the Victorians — for example, that the various parts of man's body represent the highest and final stages of which evolution is capable, by demonstrating that the human hand is more primitive than that of some other animals, its greatly advanced function over that of all other animals depending not on its more complex or highly developed structure but on its being connected to man's much more highly developed cerebral cortex. He produced reliable evidence in support of the view which he held in opposition to the Darwinians that acquired characteristics may be transmitted.

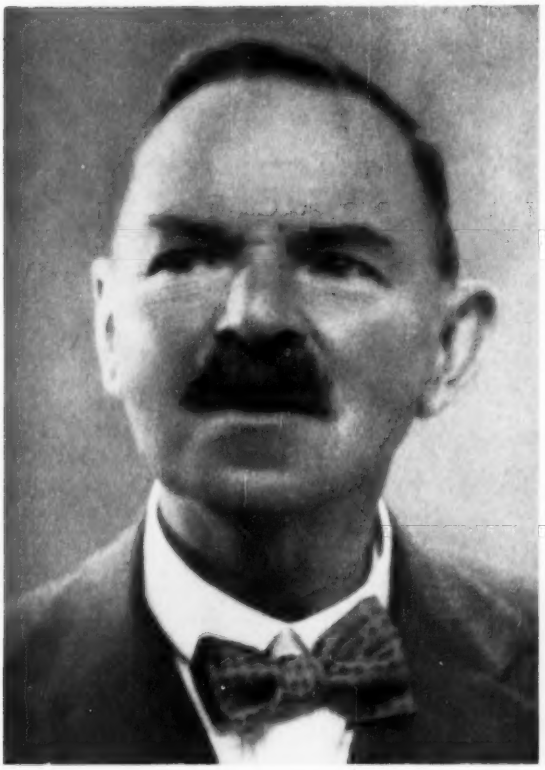
His career was marked by the rich contributions he made to biology and to its literature. He wrote as he spoke in aptly chosen words and his words had magic in them. Sir Geoffrey Jefferson described him as a " spell-binder " and told me that he had more influence on students than any other teacher he had known. He would pack his class room with students eager to hear his anatomical discourses, and some of them have been heard to say: " Of course this is no use for examinations." When students who realise this voluntarily attend lectures on anatomy I think it will be generally agreed that there is magic about.

And what of him as a man ? He was most approachable, kind and informal and had the true humility of greatness. Thirty-five years have passed since I first met him and I shall always remember his charming and lively informality. It was in the examination hall where I was a candidate at an examination in anatomy, and in reply to an answer that I had given him relating to the sex of a hip bone, he asked me whether I would bet sixpence on it. Needless to say such an approach at once put an apprehensive candidate at ease and made the oral examination so much the less formidable. Long afterwards as Chairman of the Museum and Research Committee in the College I saw a good deal of him, and these meetings, enlivened by his reports, added to the pleasure of serving on this committee.

We have lost not only one of the greatest exponents of both comparative and human anatomy of our time, but a learned philosopher who was a guide and inspiration to his students and friends all over the world.

It has been said of Wood Jones that in his lifetime he grasped and interpreted so wide a knowledge of man and his environment that few scientists could approach him for wealth of learning. Nevertheless throughout the works of Nature he perceived and appreciated a beauty of design, a basic pattern which exists throughout the realm of living things. He was a student of the Bible, which he kept at his bedside and was reading not long before he passed into the dark valley beyond which his and all our unsolved problems will be made clear.

In certain respects we may compare Wood Jones with another literary minded anatomist who lived a century before him. Oliver Wendell Holmes was also a spell-binder who delighted his classes with his whimsical teaching, likening the mesentery to the shirt ruffles of a preceding generation and the corpus collosum to the band uniting the Siamese twins; Wood Jones, less poetical and less whimsical, was nevertheless, as a prose master, no unworthy follower of the Muse, and his writings will long remain as a reminder to us that even in these days we may find science and literature in happy combination. L. R.



DAVID McCRAE AITKEN

1876-1954

With the passing of David McCrae Aitken at Mansergh, Kirkby Lonsdale, on July 9, 1954, there departed the last of that small group of pupils, consisting of Aitken, Naughton Dunn, Alwyn Smith and McMurray, whom Sir Robert Jones trained at Liverpool in the first dozen years of this century, each of whom achieved distinction in orthopaedic surgery and contributed to its great advance. These were the men that Sir Robert first gathered together for the initial staffing of the military orthopaedic hospitals at Alder Hey and Shepherd's Bush in the first world war.

McCrae Aitken was born at Singapore in 1876, the son of the Reverend William Aitken, and was educated at George Watson's College and Edinburgh University, where he graduated in arts and in 1901 in medicine; he proceeded to the Fellowship of the Royal College of Surgeons of Edinburgh in 1904 and of England four years later. After holding house appointments in Edinburgh he came south, but to a strangely different clinical climate to which he did not easily adapt himself. Of this experience he wrote: " In 1902 I came to Liverpool and became house surgeon to Robert Jones. On the first out-patient day there appeared a boy with a dreadfully swollen knee with numerous sinuses, and through my mind ran the thought

' Excision next week and probably amputation within the next three years.' The boy, however, was merely fixed up in a Thomas's knee splint, in which he was allowed to walk, and sent home with instructions to report the following week. In a few months the outward appearance of that joint became normal, and a small range of painless movement returned."

At the Royal Southern Hospital he observed procedures in the treatment of fractures, joint disease and deformity that were certainly new and exciting, however unorthodox. But their very success convinced him of their soundness, indeed compelling him to become a most ardent advocate of the principles of Hugh Owen Thomas. When he left Liverpool, to pursue a higher qualification in Edinburgh and London, it caused him no little dismay to find that the caliper had no place in teaching hospitals in the treatment of a fracture or joint disease, although thirty years had passed since the splint and its uses were described by Owen Thomas in his first published monograph.

During his earlier years in London Aitken held a house appointment at the Bolingbroke Hospital and eventually became Resident Medical Superintendent. His main interest, however, was in orthopaedic surgery, to which he entirely committed himself on election as assistant surgeon to St Vincent's Surgical Home for Cripples (afterwards St Vincent's Orthopaedic Hospital). This appointment brought him again in intimate contact with Robert Jones, who enjoyed a consulting practice in London as well as in Liverpool, and to whom he became private assistant. On the outbreak of the first world war Sir Robert stepped into the leadership in organising a nation-wide provision for the reception and treatment of limb casualties by the establishment of military orthopaedic hospitals; and he had with him no lieutenant better equipped than McCrae Aitken, who had very considerable experience of orthopaedic surgery and was thoroughly familiar with the use of the caliper and other Liverpool appliances in traumatic surgery. Aitken took an active part in the operative treatment of military casualties at Shepherd's Bush, but he soon became preoccupied with the teaching and training of young surgeons drawn from the United States and the Commonwealth; and he often deputised for his chief at hospital inspections in different parts of the country. In 1916 he gave a lecture-demonstration before the Medical Society of London on " Orthopaedic Methods in Military Surgery " in which he stressed the importance of conservative surgery and the value of rest as practised by Hugh Owen Thomas, to whose principles he adhered without deviation.

After the war he continued at St Vincent's, but he was also appointed visiting surgeon to the Shropshire Orthopaedic Hospital, Oswestry (now the Robert Jones and Agnes Hunt Orthopaedic Hospital), where he eventually succeeded Sir Robert as surgical director. During these post-war years he was very active in various scientific societies. He was well known for his patient study of scoliosis, extending over many years—a difficult field often yielding little harvest in spite of laborious cultivation. But Aitken was recognised by his colleagues as the best informed upon this disability amongst them and one who succeeded in obtaining such correction that was often denied to others. In his sustained interest and study of spinal curvature he was the counterpart in this country of R. W. Lovett of Boston. And as a contributor to the Robert Jones Birthday Volume of 1928 he wrote on Curvature of the Spine. The following year he delivered his presidential address to the Orthopaedic Section of the Royal Society of Medicine on " Respiratory Rhythm in Physiological Relation to Movement and Posture."

In 1931 Aitken delivered the fourth Hugh Owen Thomas Memorial Lecture; this honour he well deserved—there was no more faithful disciple of Thomas. He spoke on " Rest and Exercise in the Treatment of Joints " drawing on a wealth of clinical experience and radiographic illustration while paying due tribute throughout to the teaching of Owen Thomas. In 1938 he wrote a valuable monograph entitled " Hugh Owen Thomas, His Principles and Practice " (Oxford University Press). For this work Aitken had access to many private papers, photographs and case books, all of which have lately come into the possession of the British

Orthopaedic Association and are in the care of the Royal College of Surgeons. In his book Aitken contrived to give the essential teaching of Thomas with reproductions of all the splints, and those who fail to gain access to the original writings will find this monograph a most helpful substitute.

Early in life Aitken suffered from a laryngeal malady which left him with a permanent respiratory disability. Against this handicap he fought bravely but at times it was distressing to see him embarrassed by paroxysms of coughing which were beyond his control. His fresh and sturdy appearance belied what he undoubtedly suffered. This physical weakness prevented his participating at full stretch in practice and this, combined with an over-altruistic disposition, stayed the greater recognition which his great experience and ability deserved. His serene temperament and integrity were of the very substance of his being; he possessed a friendly disposition, loved the open air and was fond of sailing. He was a member of the Royal Thames Yacht Club and delighted to entertain his friends there. Many of his old pupils all over the world whom he taught at Shepherd's Bush and Oswestry will mourn his loss and feel ever grateful for all they received from him by way of teaching and example. A. R. J.

ERIC IVAN LLOYD
1892-1954

Everyone who came in contact with Eric Lloyd rapidly became his friend.

He was born in 1892, the son of J. H. Lloyd of Birmingham. He was educated at Leighton Park, Reading, a school of which he later became a Governor, and at Trinity College, Cambridge. In his youth he was a fine athlete and was allotted a half-blue for the half-mile while at the University. He proceeded to St Bartholomew's Hospital to complete his medical

education, and passed the final examination of the Conjoint Board in 1916. He promptly joined the Royal Navy and served as a Temporary Surgeon for two and a half years, most of the time in a ship at sea. On returning to London at the end of the war he held two resident appointments at " Bart.'s," house-physician and house-surgeon, and having passed his final Fellowship he became a demonstrator of anatomy for a time and was then appointed clinical assistant to both the orthopaedic and massage departments. These two appointments initiated his ever-increasing interest in orthopaedic surgery, for he had the privilege of working under that very distinguished surgeon, the late R. C. Elmslie, who was in charge of both these departments. In addition to the routine work Elmslie allotted him the by-no-means easy task of clearing out the crowd of old chronics that was clogging the massage department.

In 1923 he had the invaluable experience of crossing the Atlantic and visiting a number of clinics in Canada and the United States. On his return to London he obtained the post of house surgeon at the Hospital for Sick Children, Great Ormond Street, a hospital to which he gave devoted service for the rest of his life. He was later appointed Medical Superintendent of the Hospital, a post he held for two years, during which he gained valuable general experience of sick children, including operative surgery for emergency cases. In 1926 he became surgical registrar, and before the end of the year was appointed to the honorary staff.

Although his interest was always concentrated on the orthopaedic work, he was not actually designated Orthopaedic Surgeon to the Hospital until twenty years had elapsed. During his early training he had served as registrar at the Royal National Orthopaedic Hospital, where he gained further general experience of orthopaedic surgery. For several years he held the post of orthopaedic surgeon to the Royal Northern Hospital, a post from which he resigned in 1948, and for a time he was Consulting Orthopaedic Surgeon to the London County Council. Throughout these years, though he was acquiring an ever-increasing general experience of orthopaedics, his heart was always dragging him towards his young patients at the Children's Hospital.

He was a scrupulously careful technician, and he gave an endless amount of thought to the details of any operation he was called upon to perform. He published a useful article on the technique of operating on the knee joint. He had quite a mechanical turn of mind and designed an ingenious director to facilitate the correct insertion of a Smith-Petersen pin in the neck of the femur. This he published in this country and also in a French journal. Later he suggested a modification of the pin to prevent it from sliding out, an introducer and an extractor. His director was rather complicated and never came into general use. He also designed quite a useful tilting table for holding a patient during operation for congenital dislocation of the hip, and he suggested the use of copper strips to facilitate the removal of a plaster-of-paris splint.

With the exception of two or three early surgical papers all the articles he wrote, probably not more than twenty in all, were devoted to orthopaedic subjects. He published a second paper in a French journal, one dealing with the manipulation of joints. He also contributed to the later editions of that well known text-book on diseases of children which still bears the name of the original authors, Garrod, Batten and Thursfield. His writings, like everything else he did, were backed by most careful and conscientious study, and invariably contained sound advice. In the second world war he readily responded to an appeal for help from an emergency hospital near St Albans which was staffed by some of his friends of St Bartholomew's Hospital and was being overwhelmed with casualties from Dunkirk. Later he became an official surgeon of the Emergency Medical Service on the staff of the hospital.

As a Fellow of the Royal Society of Medicine he served as secretary of the Paediatric Section and became president of the Orthopaedic Section. He was a Fellow of the British Orthopaedic Association and a member of the Societé Internationale de Chirurgie Orthopédique et de Traumatologie.

Having been born a member of an old and distinguished Quaker family he always

remained a keen and faithful Friend. At the beginning of the first world war, before he qualified, he served for a time in the Friends' Ambulance Unit. When he went to live in that charming house he had in Hertfordshire he became an active and valued member of the Friends of Harpenden. He was a member of the local golf club and became a vice-president of the local horticultural society.

It was his deep sincerity and integrity, coupled with cheerful friendliness, which made Eric Lloyd a real friend of all he met. To his juniors he was always kind and considerate. The painstaking thoroughness with which he approached every surgical problem never failed to impress his patients or their parents, while his natural charm and kindliness soon won their hearts. They became convinced they were dealing with a man they could trust to do the utmost that surgery made possible, and they knew he would tell them the truth if complete cure was impossible. The same happy relations existed with his colleagues who, without exception, were his real friends. As a surgeon he earned their esteem and admiration for his skill, his sound judgment and for his obvious integrity and loyalty. He himself was the most severe critic of the results of his own operations. His colleagues knew they could always rely on him for valuable and ready help. More than one of them has testified to his exceptional value in committee work: on more than one occasion it was Eric who came to the rescue, and who, with a few wise words, coupled perhaps with a touch of his wit, was able to smooth out differences when discussion of a difficult problem was becoming somewhat heated. He will always be remembered with gratitude.

Keen as he was on his work he was equally devoted to his home. In 1922, shortly before he began to work at the Children's Hospital, a friend at " Bart.'s " persuaded him to go for a trip to South Africa. Fortunately for him a Miss Antoinette Marie Roux was travelling home to Pretoria in the same ship—the lady who a few years later became his devoted wife and eventually presented him with a son and a daughter.

Of Eric Lloyd it can be said with truth " His integrity stands without blemish."

H. A. T. F.

FREMONT AUGUSTUS CHANDLER
1893-1954

We have vivid memories of the firm friendships that Mont Chandler established with all his friends in Britain, especially at the time of the Joint Meeting of Orthopaedic Associations of the English-speaking world in 1952. In this British issue of the Journal we will not try to write a full appreciation of the great qualities of this great surgeon, and of all his attributes, which will be acknowledged elsewhere. But we would like to say how great a friend he was to us, personally and individually as well as collectively, in his position of authority. He was indeed our very close friend. How greatly we shall miss his wise counsels and his firm friendship. He made his mark with us and we shall never forget.

R. W.-J.

PROCEEDINGS AND REPORTS OF UNIVERSITIES COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN

BRITISH ORTHOPAEDIC ASSOCIATION

AUTUMN MEETING, 1954

The autumn meeting of the British Orthopaedic Association was held in London on October 22 and 23, 1954, at the Royal College of Surgeons of England with the president, Professor Bryan McFarland, in the chair.

In accordance with the precedent set up last year, the day before the meeting was devoted to an instructional course in orthopaedics, each lecture being followed by a discussion. The speakers and their subjects were: *Mr I. S. Smillie* (Dundee) "The knee joint"; *Mr R. G. Pulvertaft* (Derby) "The principles of hand surgery"; *Mr F. W. Holdsworth* (Sheffield) "Traumatic paraplegia"; and *Mr H. Osmond-Clarke* (London) "Cervical discs."

The imbibition characteristics of normal and abnormal intervertebral discs—*Mr N. G. C. Hendry* (Aberdeen), speaking under great difficulties from "noises off," read a paper on the imbibition characteristics of normal and abnormal intervertebral discs. He pointed out that the degree of hydration of the nucleus pulposus depended not only on osmosis, as in the annulus fibrosus, but also on imbibition, which is the characteristic power of a gel to absorb fluid into its system until a certain physical state is reached, even against enormous pressure. In the normal nucleus most of the fluid held was due to this power of imbibition, and little increase in weight followed immersion in water or saline, in contradistinction to the annulus. In the degenerate nucleus, however, a smaller proportion was held by imbibition and more from osmotic properties, and this change became more marked as the degenerative process progressed. Any failure of imbibition in the nucleus put an extra strain on the annulus.

The "slipperiness" of articular cartilage—*Mr John Charnley* (Manchester) described experiments to measure the coefficient of friction between cartilage and cartilage, and between cartilage and other substances. The first combination was about three times as slippery as ice on ice. He discussed the theories of friction and lubrication and pointed out that in joints (which move relatively slowly) the film or wedge of fluid proposed by some writers could not in fact be an important agent. In combination with cartilage acrylic resin had a lower coefficient of friction than steel, and degenerate cartilage retained its slippery properties. Nor did wiping the cartilage dry affect his results. Sclerosed bone had a high coefficient of friction and a squeaking joint after an arthroplasty was a sign of very high friction with dangerously high forces being transmitted to the stem. He stressed that in any arthroplasty sclerosed bone should always be removed and that no weight bearing should be permitted until time had been given for the metaplasia of the newly formed fibrous lining of the joint into fibrocartilage.

Symposium on Lumbo-sacral Fusion and Low Back Pain

Four speakers discussed lumbo-sacral fusion.

Mr P. H. Newman (London) gave the results in 100 personal cases done by Chandler's method. He always included the fourth lumbar vertebra, as otherwise symptoms arising at the L.4–5 level were common. At first he used autogenous iliac bone but later ribs from the bone bank, which were more convenient and shortened and reduced the severity of the operation, but gave a significantly lower fusion rate radiologically (63 per cent as opposed to 80 per cent). Symptomatically his results were satisfactory in 66 per cent of cases of spondylolisthesis and 79 per cent of cases of "instability." If bony fusion was obtained results were satisfactory in 88 per cent, but in only 53 per cent if a pseudarthrosis could be demonstrated. Complications or poor results were due to pseudarthrosis, an untreated or later disc prolapse, operation at the wrong level, inadequate after-care, a psychologically unsuitable patient, or from the operation scar. He recommended an incision curving outwards at its lower end over the ilium rather than separate incisions if iliac grafts were to be taken, to avoid division of the posterior lumbar cutaneous nerves.

Mr E. G. Shaw (Swindon) had followed up fifty-two fusions done by onlay grafting by various surgeons, only some of which had included the fourth lumbar vertebra. Radiologically 60 per cent had successful fusions and 33 per cent had failed, the rest being doubtful or the patients dead. Match-stick cortical grafts were successful in 66 per cent whereas only 40 per cent of H-grafts had fused. Twenty per cent had suffered deep vein thrombosis as a complication. In his series the use of a plaster bed had increased the fusion rate, but he also noted that 77 per cent of those in whom bony fusion had failed were satisfied with their result.

Mr C. G. Attenborough (London) had reviewed fifty cases of lumbo-sacral fusion. Of the forty-four in which follow-up was possible fourteen had fused, and thirty were unfused. Fusion rates were much the same in spondylolisthesis as in other causes of low back pain. Of the fourteen successful fusions eight had good subjective results; of the thirty unfused only ten were satisfactory. The Bosworth H-graft had been the most successful method. The fusion rate was four times greater when a plaster bed rather than a double hip spica was used, and the chance of fusion increased the longer the patient stayed in bed, and also with the youth of the subject. He recommended that all should be nursed in a plaster bed at first, that the first change of plaster should not take place for at least six weeks, that the patient should remain in bed in a hip spica for three months, and that immobilisation should not be discarded until five months after operation. Of his " very good " results seven were fused and ten were unfused.

Mr G. Blundell Jones (Exeter) described a series of ninety-six cases. In eighty fusion had been performed at one joint only, sixty-three being across the lumbo-sacral joint, twenty-two at the L4–5 joint, and the others at higher levels. The operations were of the Bosworth H-graft type. The patients had been kept in bed for two weeks and had then been allowed up in a plaster jacket for three months, often returning to light work at six or eight weeks. A surgical belt was worn for a further three to six months, during which rehabilitation was completed. Fusion occurred in ninety-three patients, and failed in two; one patient died. The results were satisfactory in 88 per cent, and 90 per cent were able to return to their previous work, which was categorised as " heavy " in twenty-four and " medium " in forty-seven cases.

The above four speakers, together with *Mr H. Osmond-Clarke* (London), *Mr K. H. Pridie* (Bristol) and *Mr N. W. Roberts* (Liverpool) then formed a panel under the chairmanship of the president for a discussion on low back pain.

The first question demanded the panel's explanation of *the physiological basis of the relief of low back pain by a plaster jacket*. *Mr Pridie* said that disc lesions produced muscle spasm and this spasm increased tension in the disc which aggravated the spasm. He thought a jacket relaxed the muscle spasm and thus broke the vicious circle. *Mr Blundell Jones* differentiated three periods when relief might occur. The first was immediate and he agreed with Mr Pridie's explanation. The second occurred after two or three weeks and was due to the relief of hyperaemia and oedema, and the third much later when root pain disappeared with the onset of true healing. *Mr Roberts* said that no one really had any idea of the explanation. He would simply put it down to " rest," which cures most things if maintained for long enough. This led to a discussion of the correct duration of plaster immobilisation—*Mr Norman Capener* (Exeter) said " until pain has been relieved for ten days." The president thought the period should be not less than three months.

The next questioner quoted a neuro-surgical opinion that " *70 per cent of operation cases where simple removal of the disc had been performed suffered a return of pain and that a fusion operation should be done as a routine at the primary operation* " What were the panel's views? *Mr Osmond-Clarke* thought it depended on the degree of instability demonstrable at operation. He preferred the body-to-body fusion technique. *Mr Pridie* said that very few needed primary fusion. He also preferred the inter-body method which he found the easiest and most successful and which allowed the patient to get up after two weeks. Both *Mr Newman* and *Mr Attenborough* spoke in favour of the same technique, but the former remarked that it was difficult and required much retraction.

" *Does lumbar fibrositis exist, and if so what is its pathology ?* " Apparently the panel were in great doubt concerning the first part of the question but recognised the entity to which the name is given. Regarding the pathology, *Mr Roberts* and *Mr Charnley* thought it was always a disc disorder of some sort. *Mr Pridie* remarked that some patients at least were merely disgruntled people in need of an outlet. *Professor Sir Harry Platt* (Manchester) said there was no clinical evidence that the syndrome was due to a disc lesion. Nodules and areas of round-cell infiltration had been found. He thought it was a collagenous disorder of some sort, but the exact pathology did not matter as long as there was no nerve involvement. *Mr A. L. Eyre-Brook* (Bristol) had no doubt that lumbar fibrositis was a real entity. If the pain was due to a disc lesion there was no tenderness in the muscles. *Mr W. E. Tucker* (London) said that tender stiff muscles were found 1) in cases of occupational or postural strain, and 2) in muscle spasm, which could result in secondary changes in the muscle itself causing persistent tenderness.

"*Could the panel explain the absolute increase in sciatica and low back pain?*" *Mr Pridie* agreed that the increase was real, and put it down to modern life in general with its lack of rest, low furniture, etc. *Mr Newman* stressed that with increased intelligence went a lower pain threshold, and he therefore thought that the present-day better standard of living might be significant. *Mr Roberts* did not believe that anyone knew if the increase was real—there was no definite evidence.

Mr G. K. McKee (Norwich), reverting to the papers on lumbo-sacral fusion, asked "*whether the disc should be explored at the same time as the fusion?*" The questioner thought it should if there were neurological symptoms or signs. He used interfacetal bone pegs. *Mr Newman* agreed; the disc should be explored if there was or had been sciatica present. *Mr Osmond-Clarke* remarked that absolute operative fixation was not everything, and *Mr Pridie* thought exploration of the disc a vital part of the operation. *The president* thought the operations should be done separately, doing one or other as the indications demanded, but *Mr McKee* said there were cases where both were needed at once. *Mr W. Sayle-Creer* (Manchester) thought that if the disc had been explored an inter-body fusion was preferable, but, if not, a Bosworth H-graft was the most satisfactory.

"*Does the panel believe in fusing one or both sacro-iliac joints?*" The panel were not sure that either should be fused. *Mr Shaw* believed that both should be fused in cases of sacro-iliac arthritis, *Mr C. H. Cullen* (Manchester) thought it was indicated in post-traumatic cases, and *Mr W. E. Tucker* remarked that true sacro-iliac strain was a very rare lesion.

"*Can a cervical disc lesion cause low back pain?*" *Mr Osmond-Clarke* thought not, though both could occur together. But a big lumbar disc prolapse could pull on the theca and cause pain higher up the back. *Mr John Charnley* (Manchester) referred to the condition he called "acute glaucoma of the disc" which could be recurrent and lead to fibrosis and later sequestrum formation in the disc. This introduced the era of mechanical failures in the disc, but he thought there was no real prolapse until pain was felt below the level of the thigh. *Mr R. H. Metcalfe* (London) described his personal experience of cervical spondylosis, and thought that the aggravation he had suffered from short-wave diathermy was due to congestion and swelling around the intervertebral foramina.

Mr F. C. Durbin (Exeter) asked for the panel's *indications for lumbo-sacral fusion*, excepting disc prolapse. *The president* thought the main indication was pain, persistent, disabling, and not relieved by prolonged conservative treatment. *Mr Roberts* thought it vital to make a diagnosis first. Ninety-nine times out of a hundred we did not know why they had pain, and therefore the operation must be a rare one. We must first be convinced that there was some truly local lesion. *Mr Pridie* thought it should be done for those patients who had a recurrence of symptoms after removal of a disc. *Mr Osmond-Clarke*, while agreeing with the president's indication, thought this should include any local instability, and recommended that more use should be made of discograms. *Mr Newman* said the indications must be based on clinical assessment, those with chronic pain or recurrent sharp attacks at short intervals being the most suitable. *Mr Blundell Jones* remarked on the difficulty of defining the correct level for fusion. He thought that the patient should have had symptoms for not less than a year, and found local infiltration and films taken during bending useful adjuncts to correct localisation. Usually one had to await the appearance of definite radiographic changes. *Mr Attenborough* asked whether one could assure the patient that operation would relieve the symptoms. He himself was doubtful of this. *Mr Shaw* said one must insist on the presence of a local lesion. The operation was useless if there was pain at other levels.

"*How high did the panel assess the psychogenic element in low back pain?*" *Mr Shaw* remarked that in his series six patients had been labelled psychogenic and all had been cured by operation. *Mr Roberts* did not think that this necessarily meant that they had had organic lesions.

"*What did the panel think gave rise to the greatest difficulty in differential diagnosis?*" *Mr Osmond-Clarke* was convinced that the telephone should take the highest place. *Mr J. R. Armstrong* pointed out that at operation 2 per cent were found to have some form of tumour within the spinal canal. We should be prepared to make more use of myelograms, but not as a routine.

Mr J. S. Ellis (Southampton) asked "*what place, if any, was there for manipulation in low back pain and sciatica?*" *Mr Roberts* thought it was criminal to manipulate under anaesthesia, except in the case of the young woman with little or nothing wrong with her. *Mr Pridie* said that life was just one big manipulation. He thought it a most valuable weapon, but one that should never be used in the presence of a tension lesion. *The president* said he found that straight distraction under anaesthesia was valuable but that other manipulations were dangerous. *Mr McKee* said that recently he had stretched all patients on a special table without anaesthesia before applying a plaster jacket in hyper extension and thought he was getting improved results. *Mr Blundell Jones*

felt that there was a place for manipulation in the later stages of treatment. *Mr Eyre-Brook* said he did not believe that a disc could be sucked back and stay back.

"*One-third of all orthopaedic out-patients complained of low backache; had the panel any suggestion for coping with this vast number?*" The panel had none and on this satisfactory note the meeting was adjourned.

Controlled movement in the treatment of congenital dislocation of the hip—*Mr J. Rowland Hughes* (Oswestry) showed a film. He had devised a modification of the Burke splint which allowed movement in the coronal plane and in which the degree of abduction of the hip could be adjusted. To the metal basis of the splint four ball castors were attached on short legs, and the film demonstrated how the child could move about the floor when face downwards by the use of its arms and controlled abduction-adduction movement at the hips.

Recovery from poliomyelitis—*Mr W. J. W. Sharrard* (London), introduced by Mr J. I. P. James, reported the result of an investigation on 140 poliomyelitis cases, each observed for thirty-six months with the object of discovering how long and to what extent gain in power of paralysed muscles might be expected to continue. This paper is published in full elsewhere in this number of the Journal.

Treatment of "frozen" shoulder by manipulation and cortisone—*Mr J. E. Buck* (Woolwich) reported the results in twenty-two patients with "frozen" shoulders whom he had treated by manipulation together with an intra-articular injection of 100 milligrams of cortisone in 4 millilitres of solution. This was followed by physiotherapy on usual lines. Twenty-one out of twenty-three shoulders gave excellent or good results. He thought his results were better than those he had obtained by other methods but admitted that this was only an impression and that he lacked controls.

The results of a re-alignment operation in the treatment of recurrent dislocation of the patella—*Mr M. Harrison* (London) discussed the results of a re-alignment operation for recurrent dislocation of the patella performed at Oxford on thirty knees, the insertion of the ligamentum patellae being transposed downwards and medially with release of the outer margin of the patella. In twenty-seven knees there was known to have been no recurrence of the dislocation, but some patients had a sense of instability. There was no case of later patello-femoral arthritis, although one-third of the patients had slight aching at the end of a heavy day. He stressed, however, that none had yet reached the age of fifty. In view of the reports of the poor condition of the patellar articular surface in cases in which the patella had been excised for this condition he wondered whether his results were an indication that some recovery might take place later. Four patients had developed some degree of genu recurvatum which he thought was due to muscle imbalance increased by later growth from the pulling-down of the often high patella; none had occurred when the operation had been performed after the age of fourteen, which he therefore believed should be the lower age limit for this type of operation.

Adaptation to adversity—*Mr J. K. Stanger and Mr J. V. Todd* (Newcastle-upon-Tyne) showed a film, to which introductory remarks were made by *Mr Gordon Irwin*. This film showed the impressive and inspiring ability of three severely crippled persons to look after themselves and to earn their living. They were: 1) a woman who had lost in childhood all her toes and both hands through the carpus from frost-bite and who earned her living as a cleaner; 2) a man who had lost both arms and one eye from war wounds, who worked as a clerk; and 3) a woman who had complete birth palsies of both arms who did everything, even fine embroidery, with her feet.

Tendon transplantation in the prevention of foot deformities after poliomyelitis in children—*Mr M. F. Pilcher* (London) assessed the results of tendon transplantation in the prevention of deformity of the foot in 147 cases of poliomyelitis in children. He divided these into nineteen cases of calcaneus, eight of calcaneo-cavo-varus, thirty of calcaneo-cavo-valgus, thirty-nine of medial cavus, twenty-nine of plano-valgus, and twenty-two of equino-varus. Of these, forty-eight were unaltered, sixty-three were improved, and thirty-six were worse. He remarked that reinforcement of a completely paralysed calf never overcomes a strong tibialis anterior, but if the latter is weak such an operation may result in equinus. The principles of operation were: 1) to reinforce the weak anatgonist, and 2) to transplant the actively deforming muscle to a site where it would be functionally useful. It was essential to correct structural deformity first and to protect the transplant for several months. The dangers lay in producing instability, over-correction, or secondary deformity. He then gave details of the various transplants used in each type of deformity.

The treatment of osteoarthritis of the hip by osteotomy—*Mr J. P. Campbell and Mr J. P. Jackson* (Harlow Wood) presented a paper on the treatment of osteoarthritis of the hip by osteotomy.

The operation done by *Mr S. A. S. Malkin* was a simple abduction osteotomy at the level of the lesser trochanter without deliberate medial displacement of the shaft. The essentials were to divide the bone and to correct deformity. Of forty-six hips so treated twenty-five gave no pain and fifteen only slight pain. This relief of pain was the most valuable result of operation and improved rather than deteriorated with the passage of time. Most patients were left with 10–30 degrees of fixed flexion, and in only eleven were there more than 40 degrees of movement. The disadvantages were 1) the long stay in hospital, 2) knee stiffness, 3) loss of flexion range (40 per cent diminished, 17 per cent increased), and 4) the difficulty of applying a hip spica on very fat patients. They now used internal fixation to allow early movements and recommended slight overcorrection to allow for the adduction which tends to recur later.

This paper gave rise to a lively discussion in which many speakers joined. Various forms of internal fixation were recommended, including the Bosworth spline. The plate recommended by *Mr Lipmann Kessel* (London) had the merits of simplicity and rigidity. Regarding the mechanism of the relief of pain, simple correction of deformity and possible alterations in the position of the upper fragment were offered as explanations. Attention was called to the frequent subsequent increase in joint space after such osteotomies.

Tuberculosis of the greater trochanter—*Mr J. C. McNeur* (Melbourne) *and Mr A. E. Pritchard* (Liverpool) reviewed thirty-eight cases of tuberculosis of the greater trochanter treated at the Royal National Orthopaedic Hospital in the last twenty-five years. They differentiated those that were primarily an osteitis and those primarily a bursitis. Most cases occurred between the ages of ten and forty. Conservative treatment had given poor results, with frequent recurrences. Operation gave better results, particularly since the advent of suitable antibiotics. Eight patients later developed a tuberculous hip joint. Seven of these had been treated by complete excision of the greater trochanter, but it was notable that many hips had not been involved until many years after operation and that all had started as a tuberculous synovitis. *Mr E. S. Evans* (Alton) remarked on the poor evidence of the association between operation on the greater trochanter and later hip involvement, and asked how in any case could one avoid doing a virtually complete excision if eradication was to be successful? *Mr R. W. Butler* (Cambridge) said he also had been depressed by the results of conservative treatment. He stressed that it was essential to do a thorough excision, particularly of the ramifications of the bursa which might extend far into the glutei and also posterior to the hip capsule, a potential source of hip infection. This excision might involve some danger to the sciatic nerve. He had even seen one case in which the process had tracked down the thigh and produced a sinus in the popliteal space.

Clinical Meetings

During the afternoon of October 22, members of the Association attended and greatly appreciated clinical and operative demonstrations given by the staffs at the following hospitals: Guy's, King's College, the London, the Middlesex, the Royal Free, St Bartholomew's, St George's, University College, the Westminster, the Hospital for Sick Children, and the Royal National Orthopaedic Hospital and Institute of Orthopaedics.

Clinical Meeting at St Bartholomew's Hospital

Arthrodesis of the hip: long follow-up—*Mr S. L. Higgs* reviewed the history of arthrodesis of the hip. Bony fusion had been achieved in thirty-four out of thirty-eight patients operated upon by him and Mr I. M. Robertson. From 1942 to 1952 they had performed twenty arthrodeses for osteoarthritis of the hip in twenty-two patients, of whom seven had been followed for two years. One operation failed, but fusion was obtained later; one patient had emigrated; the remaining five all showed bony fusion and had been examined lately. The patients could all manage standing, sitting, stairs, public transport and walking from half a mile to six miles; one was a milk roundsman and another a delivery postman. Two could not manage their shoes and socks unaided.

Displacement osteotomy for osteoarthritis of the hip: results more than ten years later—*Mr H. Jackson Burrows* had records of displacement osteotomy and plaster immobilisation, personally carried out more than ten years before, for osteoarthritis of the hip in twenty-two patients. Of the fourteen patients traced, five were dead; the surviving nine were examined eleven to eighteen years (average thirteen and a half years) after operation and assessed by Mr A. H. G. Murley and Mr E. Shepherd as to pain, stiffness, and restriction of walking. There was

no pain at all on the affected side in three patients (one of these had a left osteotomy twelve years ago, and a right cup arthroplasty two years ago; the left hip had remained painless, the right was painful). Five patients made slight complaint as follows: "a bit stiff in wet or foggy weather," "very occasional pain in damp weather," "occasional ache in wet weather," "an occasional twinge," and "moderate pain since a fall fourteen years after operation." One patient was unrelieved by osteotomy, but was cured of his pain two years later by arthrodesis. The average range of hip flexion preserved was 43 degrees. Three patients (operated upon before the practice of fenestrating the back of the plaster from above the knee to below the heel at four weeks) had difficulty with shoes and stockings, two having less than 90 degrees of knee movement. Two patients were disabled by arthritis of the opposite hip, but the other seven could walk from half a mile to six miles. Mr Burrows regarded osteotomy as the most reliable of all operations for osteoarthritis of the hip.

Arthroplasty of the elbow—*Mr S. L. Higgs* showed three patients illustrating the virtue of concomitant proximal elongation of the ulna by a sliding graft to produce a good, or even exaggerated, olecranon that gave good leverage for the triceps.

Elmslie's operation for overlooked rupture of the lateral ligament of the ankle—*Mr H. Jackson Burrows* recalled that Nilsonne (1932) treated this condition by tenodesis of the peroneus brevis (described by Gallie in 1913 for peroneal paralysis) supplemented by transfer of peroneus brevis to peroneus longus. Two years later, Elmslie (1934) devised a more anatomical fascial repair for both anterior talo-fibular [anterior], and, when necessary, calcaneo-fibular [middle] fasciculi, replacing them by loops of fascia lata passed through drill holes in fibula, talus and calcaneum. This operation received less attention than an elaboration of Nilsonne's tenodesis (Watson-Jones 1940) in which the anterior talo-fibular [anterior] fasciculus was reconstructed as in Elmslie's operation but by tendon instead of fascia. The defects of tenodesis were permanent disorganisation of the peronei and neglect of the calcaneo-fibular [middle] fasciculus, which was ruptured in those patients who needed operation most. In Watson-Jones's operation V. H. Ellis had avoided drilling the talus by passing the tendon round the ligamentum cervicis, which extends from the outer part of the neck of the talus downwards to the upper aspect of the calcaneum (Jones 1944). Mr Burrows had found this a good way of anchoring the fascial strip in the corresponding part of Elmslie's reconstruction. Examples of the excellent results of this operation were shown.

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Gallie, W. E. (1913): Tendon Fixation—an operation for the prevention of deformity in infantile paralysis. American Journal of Orthopaedic Surgery, **11,** 151.

Jones, F. Wood (1944): The Talocalcanean Articulation. Lancet, **ii,** 241.

Nilsonne, Harald (1932): Making a New Ligament in Ankle Sprain. Journal of Bone and Joint Surgery, **14,** 380.

Watson-Jones, R. (1940): Fractures and Other Bone and Joint Injuries, p. 584. Edinburgh: E. & S. Livingstone.

Results of some unusual operations for the restoration of function in the upper limb—*Mr W. D. Coltart* demonstrated the following examples. *Excision of distal half of ununited ulna* in two patients who had previously undergone grafting unsuccessfully, one for Monteggia fracture-dislocation and the other for fracture of both bones of the forearm. The cases showed that excision of very much more than one inch of the distal end of the ulna was compatible with excellent function. *Triceps to biceps transplant.* Two patients were shown in whom the whole triceps had been transplanted into the tendon of the paralysed biceps through a single medial incision. Powerful, well controlled flexion of the elbow was regained, and supination was strengthened. *Intermetacarpal iliac graft for opponens palsy.* The insertion of a large triangular block of iliac bone through an incision in the thenar web had provided an easy way of maintaining the thumb in the position of function. *Tendon transplant for opponens palsy.* The extensor carpi ulnaris, though not often used for this purpose, had proved an excellent choice, because it was a synergist and because it provided a good opposing pull. The tendon, detached from its insertion, was transferred round the ulna from back to front, and was attached to the extensor brevis pollicis tendon previously separated from its belly and transferred to the front of the wrist. The advantages of the method seemed to outweigh the disadvantages of a subcutaneous anastomosis.

Bilateral Syme's amputation—*Mr H. Jackson Burrows* showed an appliance maker of forty-three who had had a Syme's amputation of one foot at the age of eight years and the other at thirty-seven for trophic disturbances after poliomyelitis in early infancy, a supposedly unsuitable

case. The patient had made his own prosthesis till recently; his principal pastime was skating, and in the last three years he had learnt to ski.

Painful, localised sclerosis of bone—*Mr Ivor M. Robertson* showed a woman, aged twenty-eight, with tibial pain of twelve months' duration, who presented symmetrical fusiform thickening and sclerosis of the diaphysis of each tibia, narrowing the medulla. There were no evident blood changes. Wide biopsy showed osteosclerosis, from thickening of trabeculae, and also marrow fibrosis. The pain disappeared after the biopsy and was still absent a year later.

Multiple epiphysial dysplasia: patients from three generations—*Mr Edmund Shephard* showed three generations of patients from a family that included six affected individuals in four generations.

Alcaptonuric arthritis—*Mr A. J. Harrold* showed two patients. In the first, an electrician aged fifty-three, the diagnosis of alcaptonuria had been made by Sir Archibald Garrod on the third day of life. The parents were first cousins, and an elder brother had alcaptonuria. The patient had suffered from mild pain and increasing stiffness in the back for thirteen years. Intermittent left-sided abdominal pain had also been noticed. Swelling and stiffness of the knees had started about twelve months ago. The spine was fixed in a rigid thoraco-lumbar kyphoscoliosis. The left knee presented a small effusion and limitation of flexion. Radiographs showed calcified intervertebral discs, slight osteoarthritic changes in the knees, and erosion and sclerosis of the symphysis pubis. The second patient, a woman of fifty-seven, had first noticed backache when thirty-nine. Seven years later, alcaptonuria had been diagnosed in her and her brother by Dr George Graham, who had noticed subconjunctival pigmentation when she attended for some independent complaint. Although the severe backache had subsided, stiffness was pronounced and in this patient included the cervical spine. She also had pain in the pubis and in the left shoulder. Her radiographs showed, besides the calcified intervertebral discs, mottled calcification of the costal cartilages and erosion and sclerosis of the pubic symphysis. Mr Harrold demonstrated various features of the condition, including the pigmented stiffened auricular cartilages and the colour changes produced in the urine by oxidation and by adding ferric chloride.

Historic exhibits—Exhibits were displayed in the Great Hall of the Hospital illustrative of its eight centuries of history, including a minute of 1596, appointing John Izard to be Bone-setter to the Hospital. Writings and specimens were on view illustrating the contributions made to orthopaedics by Percivall Pott, James Paget, Thomas Smith, Howard Marsh, Archibald Garrod, R. C. Elmslie, and other past members of the staff.

ROYAL COLLEGE OF SURGEONS OF ENGLAND

ROBERT JONES LECTURE, 1955

Professor Bryan McFarland has been appointed as the Robert Jones Lecturer at the College in 1955.

UNIVERSITY OF OXFORD

LECTURES AT NUFFIELD ORTHOPAEDIC CENTRE

The lecture programme for the Hilary Term, 1955, includes the following: January 13, *Major L. C. Hollister, Jun.*—"Internal Splinting of Fractures"; February 3, *Dr E. G. L. Bywaters*—"The Natural History of Rheumatoid Arthritis"; February 24, *Mr J. C. Scott*—"Results of Conservative Treatment in Fractures of the Femoral Shaft"; March 17, *Professor O. Scaglietti*—"Diagnosis and Early Treatment of Congenital Dislocation of the Hip."

UNIVERSITY OF LIVERPOOL

DEGREE OF M.Ch.(Orth.)

The examination for the degree was held in December 1954. Six candidates entered and two were successful in gaining the degree. They were: Mr B. S. Jones, of Lagos, Nigeria; and Mr M. A. Vali, of Karachi, Pakistan. The external examiners were Mr H. Jackson Burrows, of London, and Mr Norman Capener, of Exeter. The internal examiners were Mr Norman Roberts, Mr Eric Wardle and Professor Bryan McFarland.

SCIENTIFIC SOCIETIES AND REGIONAL ORTHOPAEDIC CLUBS

THE BONE AND TOOTH SOCIETY

The Bone and Tooth Society held a symposium on **Vitamin D and Bone** at the Institute of Orthopaedics, Royal National Orthopaedic Hospital, London, on September 30, 1954. Professor R. A. McCance was in the chair.

Qualitative influence of Vitamin D on bone—*Sir Edward Mellanby* gave evidence to show that Vitamin D acts on end organs as well as controlling the absorption of calcium from the gut. It was possible to vary the amount of Vitamin D intake of puppies without altering the amount of calcium absorbed and the amount deposited in comparable bones. Yet these bones varied greatly in their A/R ratios (weight of ash divided by weight of dry, fat-free bone less weight of ash), the smaller amount of Vitamin D producing thicker bones with a low A/R ratio, and the larger quantity producing thinner and stronger bones with a high A/R ratio. Thus Vitamin D affected the quality of the bones formed, quite apart from its effect on calcium absorption. Calcium balance estimations could therefore be misleading as a measure of the efficiency of processes of bone calcification.

Distribution of large dose of Vitamin D—*Dr E. Kodicek* described studies carried out in collaboration with Drs Cruickshank and Armitage. One day after a single massive dose of 1 milligram ergo-calciferol, rats showed 6 per cent of the dose in the liver and other organs and 17 per cent in the faeces. The liver appeared to be the main depository. The significant amount found in the kidneys could have metabolic implications. Much of the dose disappeared and must have been broken down into inactive products. More recently, paper-chromatography had been used to follow the fate of graded doses given to rachitic rats. After one day, Vitamin D increased in liver, kidney and faeces. On the second day, the liver stores dropped 50 per cent. Vitamin D estimations were also done on faeces of patients suffering from osteomalacia and steatorrhea, and of one normal individual, who were receiving 20 milligrams ergocalciferol daily for a number of days. No Vitamin D could be detected; this suggested that less than 1 per cent of the dose was present in the faeces. This finding was in marked contrast to the findings in rats.

Effect of Vitamin D on renal reabsorption of phosphorus—*Dr C. E. Dent*, with *Dr J. Anderson* and *Dr B. Senior*, recalled that earlier experiments on the effect of Vitamin D on renal reabsorption of phosphate had produced contradictory results. There was even more doubt whether any of its claimed actions on the kidney were direct or mediated by some influence on the parathyroids. In studies on patients with bone changes of rickets or osteomalacia and in which the biochemical changes resembled those of Vitamin D deficiency, a technique of phosphate infusion had been employed enabling phosphate clearances, glomerular filtration rate and tubular reabsorption to be determined at plasma phosphate levels of 3 to 13 milligrams/100 millilitres. Preliminary results confirmed that the kidney tubule has a maximum rate of phosphate reabsorption and that when patients were treated with Vitamin D, phosphate reabsorption increased markedly. Sometimes this increase of phosphate reabsorption occurred without appreciable change in plasma calcium level. If, therefore, the kidney tubule were influenced via the parathyroids, then it was likely that the parathyroids were controlled by some factor other than plasma calcium level. More simply one could suppose that Vitamin D acted directly on the kidney tubule, but, if this were so, difficulties would arise in explaining previous work on patients with hypoparathyroidism.

SOUTH-EAST METROPOLITAN REGION ORTHOPAEDIC CLUB

A meeting was held at Pembury Hospital on October 2, 1954. *Mr J. H. Mayer* showed twenty-three cases, all of considerable interest, so much so that time for discussion proved unfortunately rather short.

These cases included, first, a group of three cases of epiphysial foci of tuberculosis in the knee. The first, treated by immobilisation, antibiotics and repeated aspirations, showed healing with a full range of knee movement. The second had been treated similarly except for an exploration of the cavity, followed after an interval by a second exploration and packing of the cavity with cancellous bone chips mixed with streptomycin and P.A.S., followed still later by a fusion of the outer half of the femoral epiphysis to correct an increasing genu varum. Full knee movement and a discrepancy in length of only three-quarters of an inch were achieved. The third was treated by fusion of the epiphyses with preservation of normal epiphysial growth, with resulting disparity in length of one inch, which had disappeared in the two years since operation.

A case of congenital pseudarthrosis of the tibia treated by through-bolted double onlay grafts, taken from the father's shin, had resulted in sound bony union in normal alignment. There

was no local histological evidence of neurofibromatosis, though the child has café-au-lait patches and his mother has frank generalised neurofibromatosis. His sister also has café-au-lait patches.

Next there were a group of three cases of polyarthritis. The first seemed to a majority of members to be probably gouty. The second was thought to be a rheumatoid arthritis in a subject of psoriasis. The third was probably a true psoriatic arthropathy.

A group of arthroplasties of the hip included a girl of twenty-five, suffering from rheumatoid arthritis since the age of seven, who had had a Stamm arthroplasty of one hip and a Batchelor arthroplasty of the other. Function of both hips was excellent and the good stability of the Stamm arthroplasty, despite a radiographic appearance strongly suggesting extreme instability, was a subject for comment. An excellent functional result in a bilateral Smith-Petersen cup arthroplasty was thought by some to be related especially to the satisfactory preservation of the length of the femoral neck on each side. A case of old unreduced congenital dislocation of the hip was shown after Smith-Petersen cup arthroplasty followed by rotation osteotomy of the upper femoral shaft.

A patient with rheumatoid arthritis had been treated by modified Judet arthroplasty of the right hip using an all-metal prosthesis, and arthroplasty of the left knee. For the latter a new prosthesis had been used which converted the joint into a simple hinge by a metal prosthesis fitted to the lower end of the femur after excision of its articular surfaces. The lateral ligaments of the joint had been preserved to achieve stability. Mr Mayer reported encouraging results with the use of this prosthesis in a total of six cases done recently and he demonstrated the prosthesis and described the method of operation.

A case of slipped femoral capital epiphysis illustrated the unusual complication of an irreducible over-reduction after the gentle use of the Leadbetter manoeuvre to reduce an acute slip. Open reduction was undertaken as a preliminary to nailing, and the child, aged eleven, had regained normal function of the hip, with no radiographic evidence of avascularity of the head fragment.

Mr P. L. Richardson showed an ingenious modification of the usual cassette tunnel for taking pelvic radiographs on the orthopaedic table, by means of which a lateral exposure could be made underneath the drapes, and which allowed the operator to stand clear of the rays during exposure.

Dr A. B. Wayte, of the Radiotherapy Department of Guy's and Pembury Hospitals, demonstrated radiographs showing the regression of osseous secondaries from a carcinoma of the breast treated by androgens with complete restoration of normal bony structure where the bone appeared to have been completely destroyed.

NORTH-EAST METROPOLITAN ORTHOPAEDIC CLUB

A meeting was held at the London Hospital, Whitechapel, on November 6, 1954, under the chairmanship of Mr H. Osmond-Clarke. Cases shown by Mr Osmond-Clarke, Mr O. J. Vaughan-Jackson and Mr Alexander Law included the following. 1) A child of thirteen months with bilateral dislocation of the knees and hips. After discussion, the diagnosis was thought to lie between gargoylism, Morquio's disease, and arthrogryposis; treatment of the knees should consist of wedging plasters, possibly followed by open reduction. It was agreed that the hips should be left untreated. 2) A case of bilateral dislocation of the patella. 3) Flail shoulder in a boy of seven years. Cases of avascular necrosis of the lunate, post-traumatic arthritis of the wrist, and osteochondritis dissecans of the elbow were also shown.

The meeting finished with an interesting colour film of the Putti-Platt operation for recurrent dislocation of the shoulder, made for Mr Osmond-Clarke by Mr R. Ruddick of the Photographic Department.

A.B.C. CLUB

The A.B.C. Club members comprise all the British and Commonwealth surgeons who have visited the United States of America and Canada, and the American and Canadian surgeons who have visited Great Britain, under the reciprocal scheme of Travelling Fellowships sponsored by the American and British Orthopaedic Associations.

On October 20, 1954, members met at a dinner at the Royal College of Surgeons of England, at the invitation of the president of the club, Sir Reginald Watson-Jones. A warm welcome was given to four new members—D. M. Brooks of London, G. P. Mitchell of Edinburgh, H. J. Richards of Oswestry, and J. N. Wilson of Cardiff. The presence, as a guest, of Dr James A. Dickson of Cleveland caused much pleasure, particularly in view of the work that he did in furthering the idea of travelling fellowships when he was president of the American Orthopaedic Association. The president read letters from Lord Webb-Johnson and from Dr R. I. Harris, both regretting their inability to be present. Many messages of greeting were received from American and Canadian members of the club.

1954 Dinner of the A.B.C. Club in the new Council Room of the Royal College of Surgeons of England. *Left to right: Back row:* H. J. Richards, Marion A. Pearson, D. M. Brooks, Dr James A. Dickson, a past-president of the American Orthopaedic Association, Sir Reginald Watson-Jones, president of the A.B.C. Club, F. G. St C. Strange, J. N. Wilson, I. S. Smillie, J. S. Ellis, P. H. Newman, J. T. Fairbank. *Front row:* James Wishart, J. C. Adams, James Patrick, G. P. Mitchell, John Charnley, Professor Bryan McFarland, president of the British Orthopaedic Association, E. C. Durbin, E. W. Somerville, J. I. P. James.

After dinner superb colour slides illustrating the 1954 tour in America and Canada were shown by Mr G. Mitchell and Mr H. J. Richards; and members were delighted to see many of the well known personalities of American orthopaedics on the screen. There was universal agreement among all who spoke that great benefit to orthopaedics in the English-speaking world derives from these travelling fellowships, and that every member should do all in his power to ensure the continuation of the scheme.

CANADA

CANADIAN ORTHOPAEDIC ASSOCIATION

ARRANGEMENTS FOR 1955 MEETING

The arrangements for the annual meeting of the Canadian Orthopaedic Association in June are as follows. A clinical day will be held in Toronto on Saturday, June 25, immediately after the combined meeting of the British, Canadian and Ontario Medical Associations. Members and visitors will then proceed to the Seigniory Club, Province of Quebec, either by car or train, where the meeting will continue from June 27–30. Any members of the Orthopaedic Section of the British Medical Association attending the combined meeting in Toronto are cordially invited to attend the meeting of the Canadian Orthopaedic Association and to take an active part in the programme. Papers from members of the British group will be welcome. It is requested that those wishing to present a paper should submit the title and a brief abstract to Dr J. Gordon Petrie, Chairman of the Programme Committee, Suite 12, 1374 Sherbrooke Street West, Montreal, Province of Quebec.

The Canadian Orthopaedic Association hopes that as many members of the British group as possible, with their wives, will find it possible to attend the meeting. Transport will be provided for them from Toronto to the Seigniory Club and everything possible will be done to assist them in obtaining suitable accommodation both in Toronto and at the Seigniory Club.

EAST AFRICA

ASSOCIATION OF SURGEONS OF EAST AFRICA

ANNUAL CONFERENCE, 1955

The Association of Surgeons of East Africa met at Makerere College, Kampala, in Uganda, from February 2–5, 1955. The Conference was opened by His Excellency the Governor of Uganda in the Main Hall of Makerere College and the presidential address followed.

Thereafter there were clinical demonstrations in the Mulago Hospital and in the Medical School of Makerere College, and a symposium was presented in conjunction with the Uganda Branch of the British Medical Association. The Annual Golf Competition was held on February 5 and there were visits to places of interest around Kampala, including a visit to the Owen Falls Dam.

RUSSIA

ACADEMY OF MEDICAL SCIENCES OF THE U.S.S.R., LENINGRAD

COMBINED SANATORIUM, ORTHOPAEDIC, AND SURGICAL TREATMENT OF SKELETAL TUBERCULOSIS

A report has been received, through the Soviet News, of the present trends in the treatment of skeletal tuberculosis in Russia. The paper has been prepared for this Journal by Professor P. G. Kornev, a member of the Medical Sciences of the U.S.S.R. and Director of the Leningrad Institute of Tuberculosis and Bone and Joint Diseases.

Professor Kornev recalls that an effective organisation for the treatment of skeletal tuberculosis was lacking in Russia before the time of the Revolution. Treatment at the few private country hospitals was accessible only to the privileged few, and the many thousands of ordinary patients with extensive disease were regarded as hopeless chronics and received practically no treatment at all. In 1936 the Soviet Government made provision for the in-patient hospital treatment of all patients with skeletal tuberculosis for so long as was necessary, and country hospitals and sanatoria were established in many parts of the country. At the same time the Leningrad Institute of Surgical Tuberculosis and Bone and Joint Diseases was created to co-ordinate the work of all the bone tuberculosis institutions, to train specialists, and to conduct clinical and laboratory research in this and allied fields. In consequence partly of these administrative measures and partly of improved methods of treatment, a striking reduction in the incidence of severe destructive disease has been observed and the prognosis has become much more favourable.

Research work—Extensive clinical and pathological studies have been undertaken to determine the pathogenesis and evolution of tuberculous lesions of the larger joints, especially the hip and the knee; and it is claimed that "experimental models" have been obtained in rabbits and dogs of a chronic specific bone lesion which under certain conditions passes on to the joint and to soft tissues, as in man. "We now successfully use the artificially induced tuberculous areas in bones as models for experimental therapy conducted under radiological, bacteriological and histological control."

Pathology of joint tuberculosis—On the basis of this research, Professor Kornev concludes that tuberculous inflammation of a joint of haematogenous origin begins first in the bone adjacent to the joint, and not in the synovial membrane. Isolated tuberculous foci are formed in the spongy bone of the epiphysis and metaphysis—a tuberculous osteitis or osteomyelitis. This process develops slowly and insidiously without causing noticeable symptoms. This, the pre-arthritic stage, is the first stage of a three-phase evolution of tuberculous arthritis. In the second or arthritic stage, there is inflammation of the joint tissues proper. The third, or post-arthritic stage, is occupied by gradual walling-off and repair.

Much importance is attached to nervous reflex influences proceeding from the primary focus in the bone. "The same nervous reflex influences, which later issue from the diseased joint, explain the characteristic dystrophic changes that occur in the bones (osteoporosis, destruction, disturbance of growth) and in the contiguous muscles (contracture, atrophy), and also the vegetative disorders which are so frequent in cases of joint tuberculosis. The longer the source of nervous receptor irritations continues to exist the greater is their influence on trophic functions and on the persistence of dystrophic changes."

Treatment—The importance of early treatment is stressed, and an improvement in diagnostic facilities is making early treatment possible in an increasing proportion of cases.

The mainstay of treatment is by a combination of constitutional measures (sanatorium regime, with antibiotics) with local immobilisation of the affected joint. But operation is increasingly being recognised as a valuable adjuvant, especially in preventing recrudescence of the disease.

The use of antibiotics—Streptomycin is the main antibiotic drug in general use. It was introduced experimentally at the Institute in 1951, and detailed studies have been carried out in about 500 cases. Streptomycin is believed to be of most value in the treatment of general complications—meningitis, miliary disease, lung disease—and on local complications such as chronic discharging sinuses. Consequently it has led to a reduction of mortality. But except in early lesions (especially in children) it did not seem to have a striking influence on the local focus in bone or joint. "Streptomycin has not altered the situation to any great extent; it has not disturbed the cycle of development of the process and has rather extended than narrowed the indications for operation."

Operative treatment—Under modern conditions operations on tuberculous joints are regarded as reasonably safe, provided they are carried out in the quiescent stage and not during the phase of activity. In most cases the aim of operation is to eliminate the focus of infection by excision of diseased tissue. Excision with arthrodesis is recommended for the knee, and a similar method is used for the hip. When a direct attack on the tuberculous lesion is considered undesirable or technically impossible resort is had to extra-articular arthrodesis. In cases of isolated extra-articular bone disease Professor Kornev advocates excision of the lesion to prevent spread of infection to the joint. Antibiotics—penicillin and streptomycin—are used after operation to prevent suppurative complications.

Results—Remarkable improvements in the results have been recorded in recent years. For instance, the general mortality has been reduced within a decade from 8 per cent to 1·1 per cent. At the same time the incidence of sinuses has been greatly reduced, and the former large category of "hopeless" chronics is beginning to disappear. Moreover the recurrence rate has been much reduced, thanks largely to operative excision of the lesion when quiescence has been reached.

Professor Kornev is the author of a monograph entitled *Bone and Joint Tuberculosis: Principles of Pathology, Diagnosis and Treatment*, first published in Moscow in 1951.

Book Reviews

DEGENERATIVE CHANGES OF THE SHOULDER JOINT AND THEIR CONNECTION WITH SHOULDER PAIN. By Olof Olsson. 9½ × 7 in. Pp. 130, with 63 figures and 30 tables. 1953. Göteborg: Acta Chirurgica Scandinavica, Supplement 181. Price Sw. cr. 15.

The clinical examination of 828 patients and the dissection of 106 shoulder joints of those who subsequently died is a monumental task. From this the author has amassed a great number of facts concerning the relation between shoulder pain and degenerative changes in the joint. His findings will serve to emphasise our present knowledge of a number of points, for instance that soft-tissue disruption often occurs "silently" and with increasing frequency with advancing age—up to 75 per cent of examined specimens in his oldest group—and that cardiac disease is a more common cause of shoulder pain than is generally supposed: in his series shoulder pain occurred almost twice as frequently among patients with evidence of heart disease. The term "shoulder joint" is used by the author to embrace the gleno-humeral joint, subacromial bursa and biceps tendon, but the functionally inseparable acromioclavicular joint is unaccountably excluded and was not examined in his morphological preparations. This may explain the lack of association found in this study between degenerative changes and a history of shoulder pain.

The close relation between chronic rupture of the rotator cuff and radiological irregularity of the greater tuberosity is instructive:

TABLE 14

Frequency of Cuff Ruptures and of Roentgenologically Visible Tubercular Irregularities

	Irregular tubercles	
	+	−
Cuff rupture +	40	5
Cuff rupture −	10	46

Fig. 57

Fig. 58

There are few conditions in which a disciplined analysis of clinical signs and symptoms is so rewarding as in the differential diagnosis of shoulder pain, and it is therefore to be regretted that the author has not served the *digestif* of a more balanced assessment with his splendid feast of facts.—A. W. Lipmann Kessel.

THE SHELF OPERATION. An Evaluation of Results in Congenital Dysplasia, Subluxation and Dislocation of the Hip Joint. By Åke Jakobsson. From the Orthopaedic Department of Karolinska Institutet, Stockholm. 9 × 6 in. Pp. 120, with 34 figures and 22 tables. 1954. Acta Orthopaedica Scandinavica, Supplement 15. Copenhagen: Ejnar Munksgaard. Price D. kr. 18.

Anyone interested in congenital dislocation of the hip would do well to read Åke Jakobsson's study of the part that the shelf operation may have in treatment. His contribution is clear, concise and comprehensive. The origin of the operation in 1891, its subsequent desuetude and its revival are all graphically described. Two hundred and thirteen operations are reviewed and analysed. One hundred and sixty-seven patients were involved, and the author is to be congratulated on the number he could trace. One fancies that he must deal with a more static populace than ours. He assesses the results particularly in relation to whether the patients had been treated for congenital dislocation before (" secondary cases "), or whether the operation was the first attention the patient received (" primary cases "). He also relates the results to age, and to the initial indications for the operation. The radiographs are clear. There are some very interesting diagrams and models, a most useful discussion of the problems of surgical technique, and some well considered conclusions. The whole work is characterised by a frank and reliable touch which makes it all the more commendable.—Bryan McFarland.

THE EFFECT OF CORTISONE ON THE FORMATION OF TENDON ADHESIONS AND ON TENDON HEALING. An Experimental Investigation in the Rabbit. By Nils Carstam. 9½ × 7 in. Pp. 111, with 23 figures and 28 tables. 1953. Stockholm: Acta Chirurgica Scandinavica, Supplement 182. Price Sw. cr. 15.

This is a constructive work which will well repay study, both on account of the author's observations and conclusions, and because it is a fine example of carefully controlled research. Carstam points out that " in traumatic and reconstructive surgery one of the main problems is the restoration of the gliding action of a tendon after injury." He sets out to investigate the influence of cortisone in the rabbit upon the healing of tendon, and upon the development of adhesions after tendon injury. He establishes the fact that cortisone has a marked suppressive effect upon adhesion formation and does not cause appreciable delay in healing of a divided tendon, provided retraction of the tendon ends is prevented. Carstam concludes that cortisone may therefore be of practical value in man, particularly for the prevention of adhesions after tenolysis with tendon continuity maintained.—R. G. Pulvertaft.

FRACTURAS DE LA EXTREMIDAD SUPERIOR DEL HÚMERO. By Carlos E. de Anquín, University of Cordoba, Argentina. 9½ × 6½ in. Pp. 148, with 61 figures. 1954. Cordoba: Lutz, Ferrando y Cia, S.A.

Dr Anquín, a surgeon of the Argentinian Army, whose volume on painful conditions of the shoulder was reviewed in this Journal (**36-B**, 347) last year, has written now on the disorders of the shoulder associated with fractures. After an historical review, mainly of the methods for classification of these fractures, he offers his own nomenclature integrating some of the previous classifications, the main groups being intra-capsular fractures, fractures across the tuberosities, fractures of the surgical neck and complicated fractures. The clinical study is based on personal experience, a fact which gives this little book a particular interest. The last chapter describes substitution of the head of the humerus by an acrylic prosthesis, of which a model designed by the author is described. Three cases operated by this method are mentioned. Of these three cases only the last one seems to us to justify the procedure. The book is well illustrated and may be of help to those who can read Spanish.—J. Trueta.

PERTE DE LA FLEXION DU COUDE: TRAITEMENT PAR L'OPÉRATION DE CLARK-SEDDON. By François Pichat, aide d'anatomie à la Faculté de Lyon. 9½ × 6¼ in. Pp. 63, with 4 figures. 1954. Lyon: Bosc Frères. Copies not for sale; obtainable from the author on request.

This brief thesis—it contains less material than the number of pages suggests—is concerned with J. M. P. Clark's pectoral transplant for paralysis of the muscles that flex the elbow. Clark's original paper is quoted at length, and the modifications of Seddon and other surgeons are described; the indications for the operation and the anatomy of the region are discussed in some detail. Seddon's results with seventeen patients are quoted, and seven more, operated on by members of the surgical staff at Lyon, are added. The latter were all performed for brachial plexus

injuries and in six instances arthrodesis of the shoulder was carried out as well. The results were good or excellent in five of the seven.

This publication is useful because it proclaims an operation that should be more widely known. It is one of the most successful of all muscle transplants and a really valuable addition to the repertory of orthopaedic operations. Its inception is due to the inventive genius of Mr Clark, and if eponomy is to remain of value, his name should appear alone in the credit title. I am sure that Mr Seddon, who was one of the earliest to appreciate the value of the operation, and who has practised it so often and so successfully, would be the first to agree.—Philip WILES.

INTRODUCTION A LA PRATIQUE CHIRURGICALE DE LA HANCHE. By Jean CHEVNEL. 9½ × 6 in. Pp. 104, with 27 figures. 1954. Paris: G. Doin & Cie. Price 975 fr.

This monograph is one of a type rarely published in England. It consists largely of a summary of various aspects of the pathology and modern surgery of the hip joint. The author has carried out some personal work on the blood supply of the head and neck of the femur and of the round ligament. In this chapter he repeatedly makes gracious acknowledgment of the work of Harrison and Trueta; indeed he warmly refers to the latter as "le Maître d'Oxford." The author has also worked on the comparative anatomy of the hip joints of newly born children in the School of Medicine at Dakar, where congenital dislocation of the hip is rarely found in the indigenous population. From examination of twenty-five post-mortem specimens he concludes that at birth the African femoral neck has much less anteversion and the acetabulum a greater depth than is found in European infants. This is a different point of view from the opinion frequently heard in South Africa that congenital subluxation in the hip in the Bantu is given correct abduction treatment from soon after birth by reason of the way the infants are carried on their mothers' backs. These matters are not discussed at great length, and anybody who has retained a schoolboy knowledge of the language can easily follow the simple arguments and diagrams.—K. I. NISSEN.

BASIC ANATOMY. By G. A. G. MITCHELL, *O.B.E.*, *T.D.*, M.B., Ch.M., D.Sc., Professor of Anatomy and Director of the Anatomical Laboratories in the University of Manchester, and E. L. PATTERSON, M.D., B.Sc., Senior Lecturer in Anatomy in the University of Manchester. 10 × 7 in. Pp. viii + 438, with 286 figures, some in colour. Glossary and Index. 1954. Edinburgh and London: E. & S. Livingstone Ltd. Price 45s.

This is an excellent book for undergraduates and others. It is not a book of detailed anatomy and yet is more than a bird's eye view. In their preface the authors state that their chief aims are: *a*) to present basic information about human anatomy, and to synthesise facts from various subjects into a story that may serve as a bridge between biology, anatomy, physiology and clinical subjects; *b*) to attempt to explain briefly how man has evolved and developed, how his various cells and tissues are arranged, and *c*) to provide an epitome of the main facts about the various bodily systems. In these and other aims they have been singularly successful and have produced a book which is informative, thought provoking, and eminently readable. A glossary is provided for those whose classical vocabulary is hazy. The illustrations—line, microscopic and photographic—have been well chosen and clearly produced. Altogether it is, within its field, a book to be commended confidently.—Robert I. STIRLING.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY. By Hamilton BAILEY, F.R.C.S., F.A.C.S., F.R.S.E., Emeritus Surgeon, Royal Northern Hospital, London. Assisted by Allan CLAIN, M.B., F.R.C.S., Senior Surgical Registrar, Royal Cancer Hospital, London. Twelfth edition. 9 × 6 × in. Pp. xii + 456, with 681 figures, many in colour. Index. 1954. Bristol: John Wright & Sons Ltd. Price 38s. 6d.

Although Allan Clain has assisted in the preparation of this edition he has not been allowed to alter much. This is right and proper, for otherwise the book would lose its character. Nostalgia forbids that any one of its well known pictures should be omitted; yet so great a number gives ample opportunity for carping criticisms. For example, we suggest omitting Figures 315 and 681: Figure 315 because tapping the middle metacarpal for a fracture of the scaphoid is not a method of diagnosis to instil into a student; and Figure 681 because, on account of its rarity, a student would be better not to know of the existence of a psoas bursa. For over twenty-five years every student on entering the clinical period has bought a stethoscope and a "Hamilton Bailey." Who would like to decide which is the more essential?—George PERKINS.

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IMPORTANT ANNOUNCEMENT

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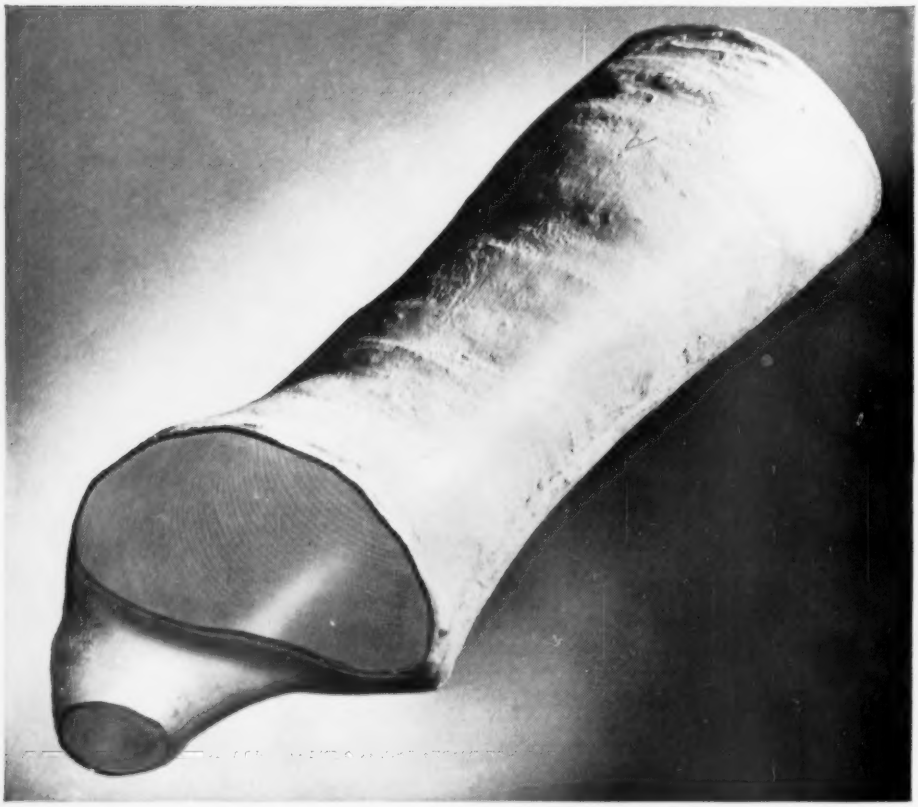

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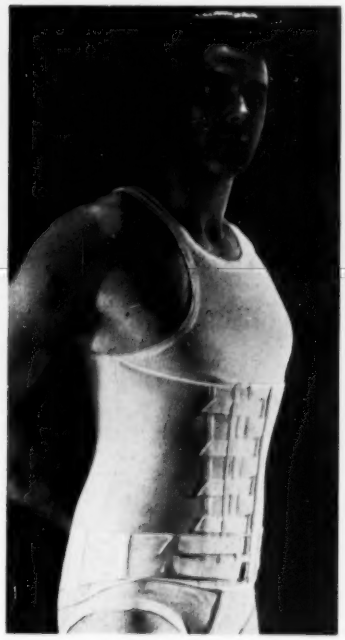

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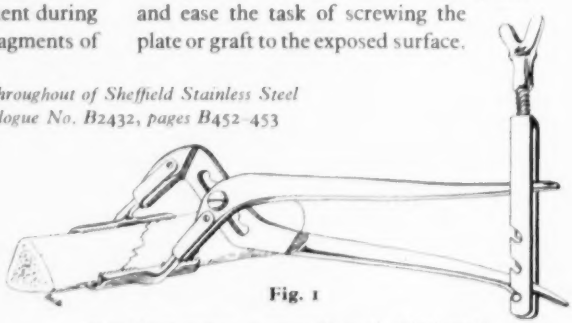

Fig. 1

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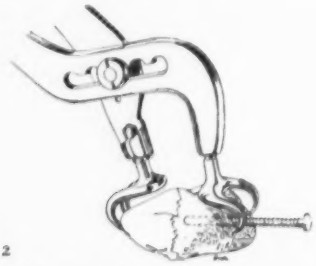

Fig. 2
Hooked jaws used to grip a fractured patella.

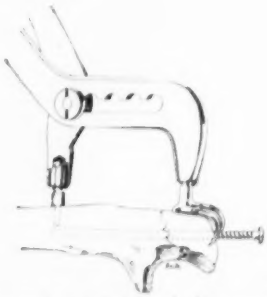

Fig. 3
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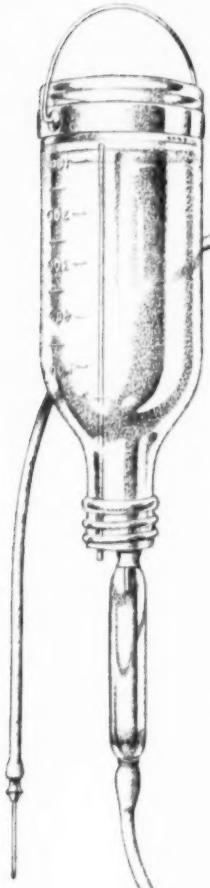

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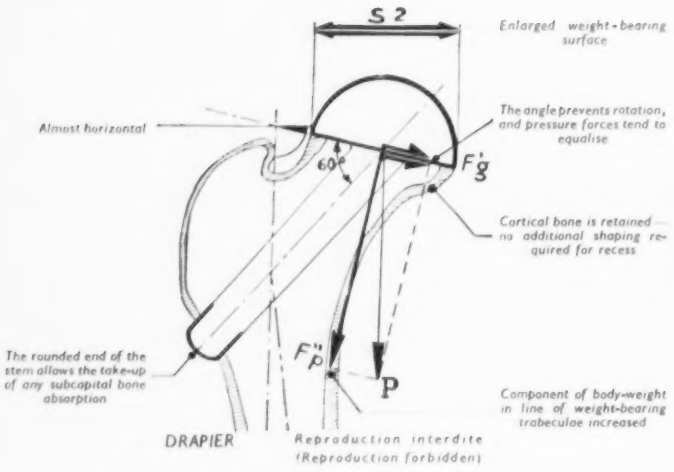
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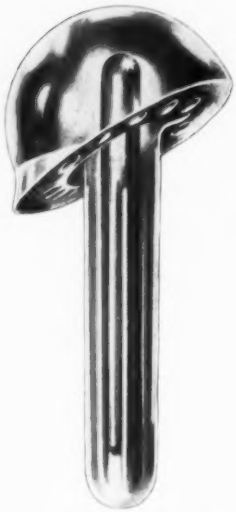

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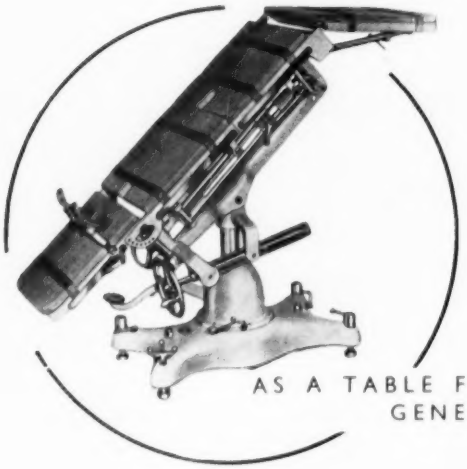

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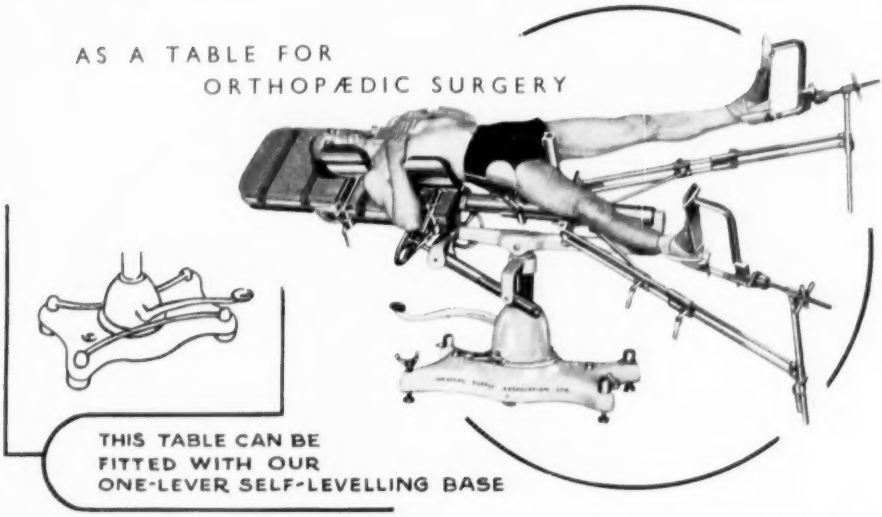

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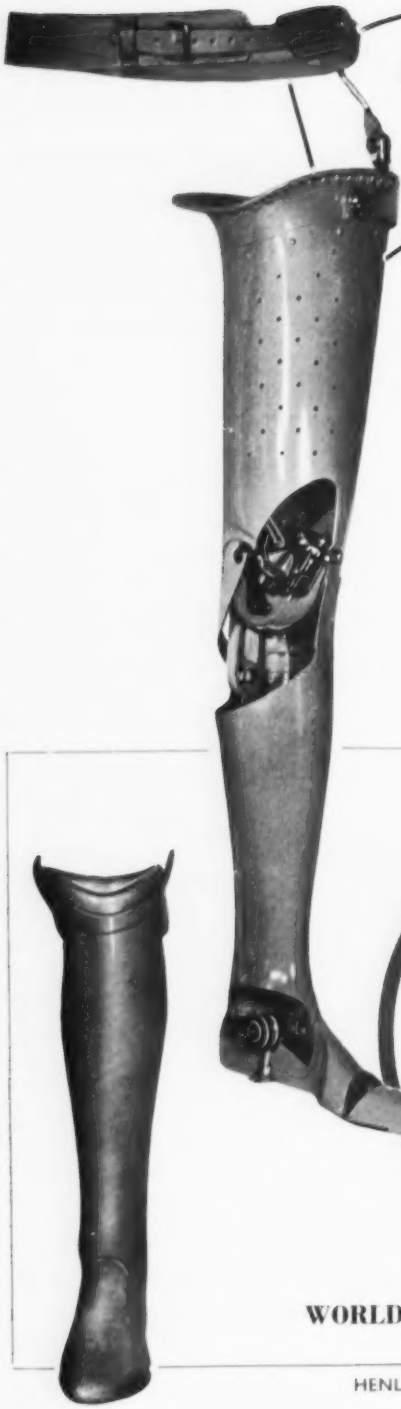

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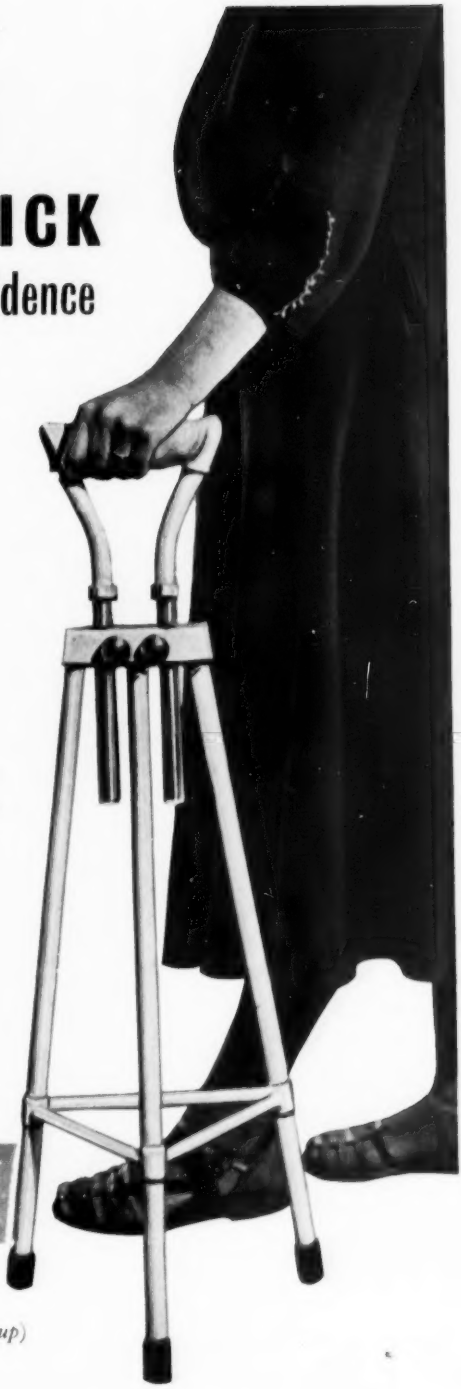

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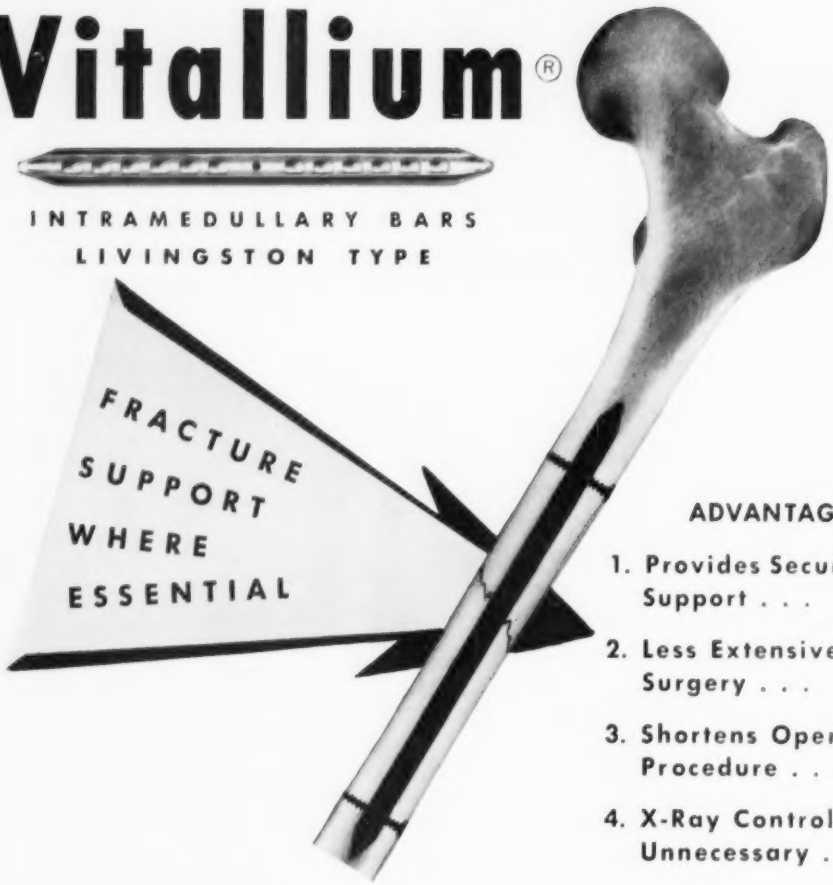

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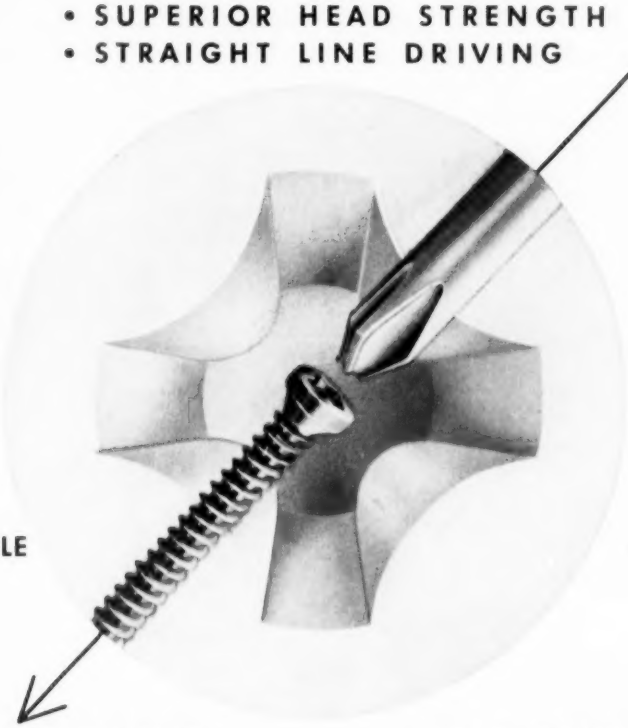

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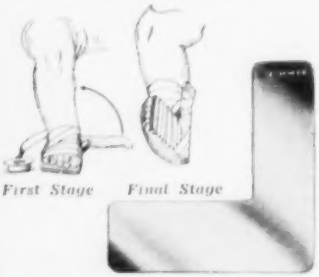

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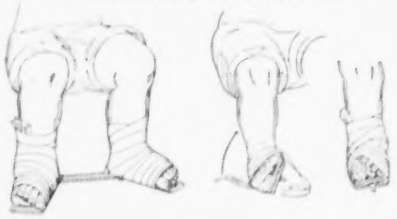

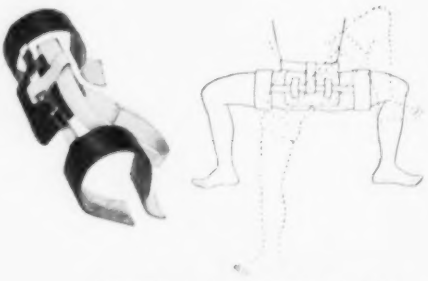

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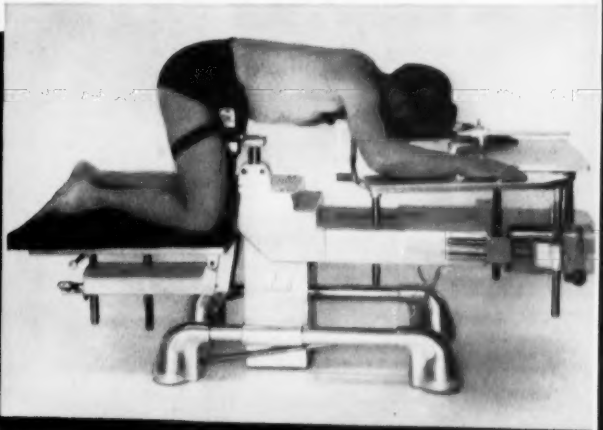

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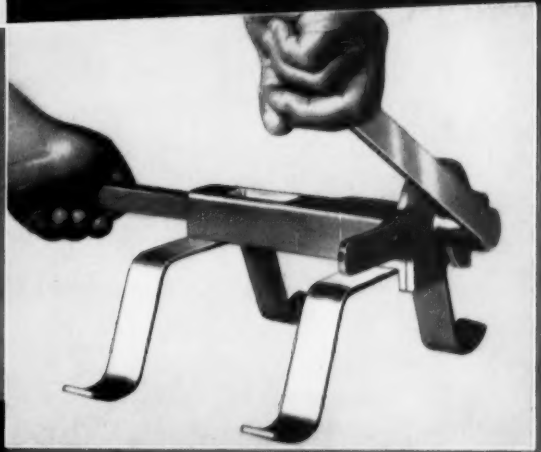

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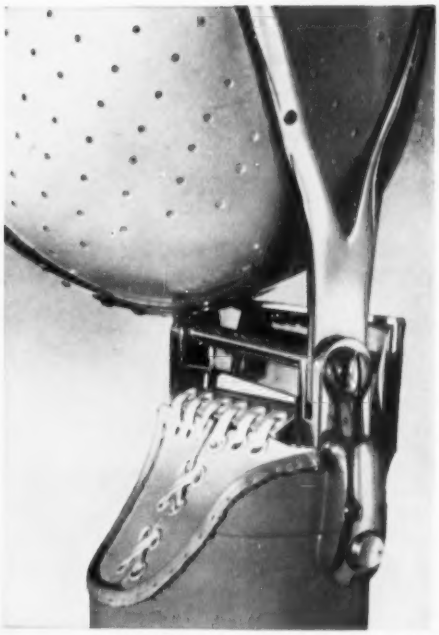

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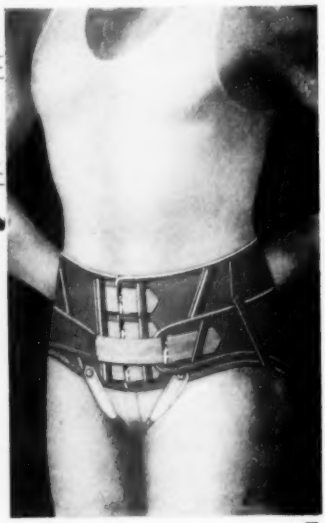

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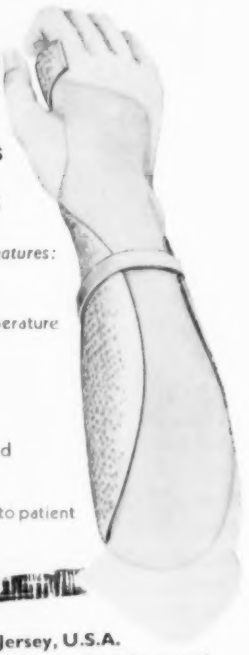

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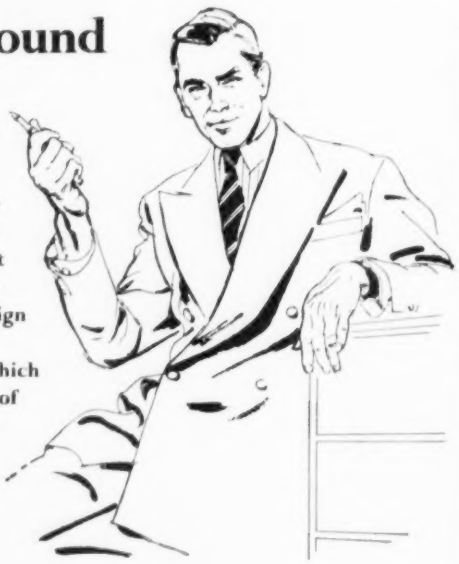

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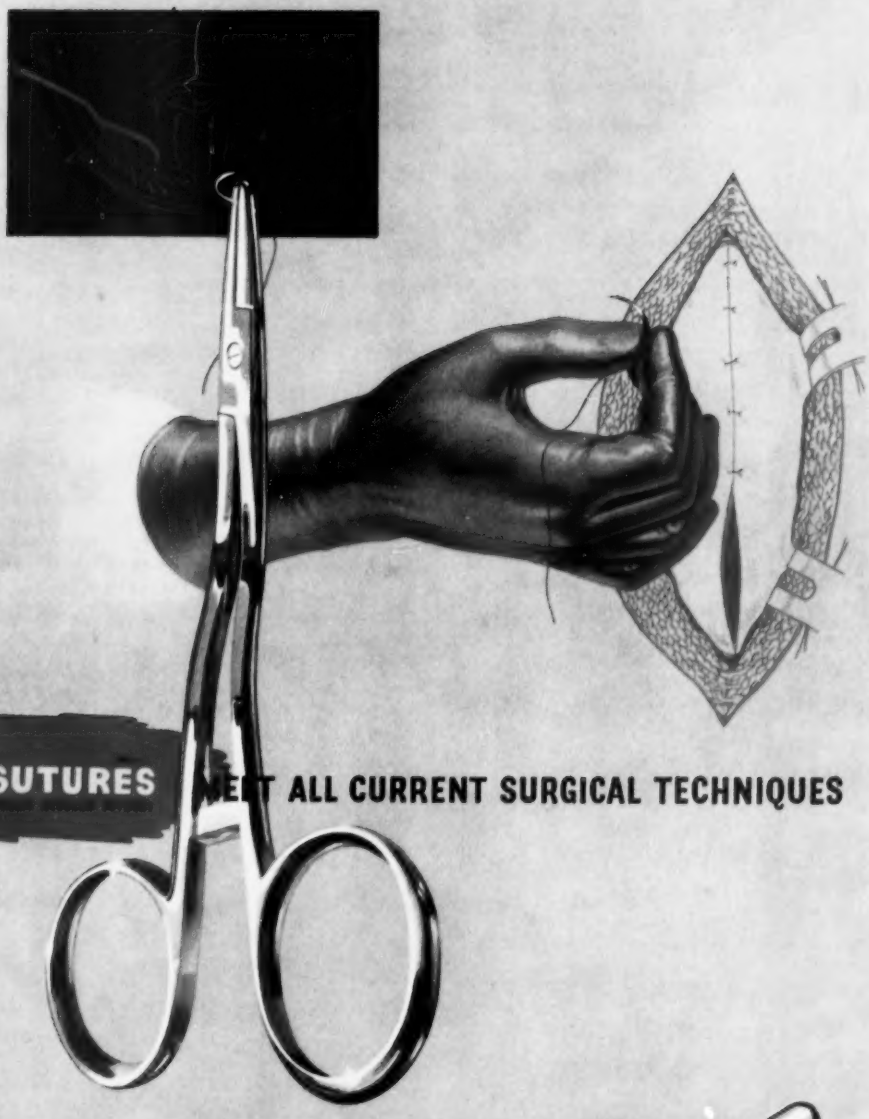

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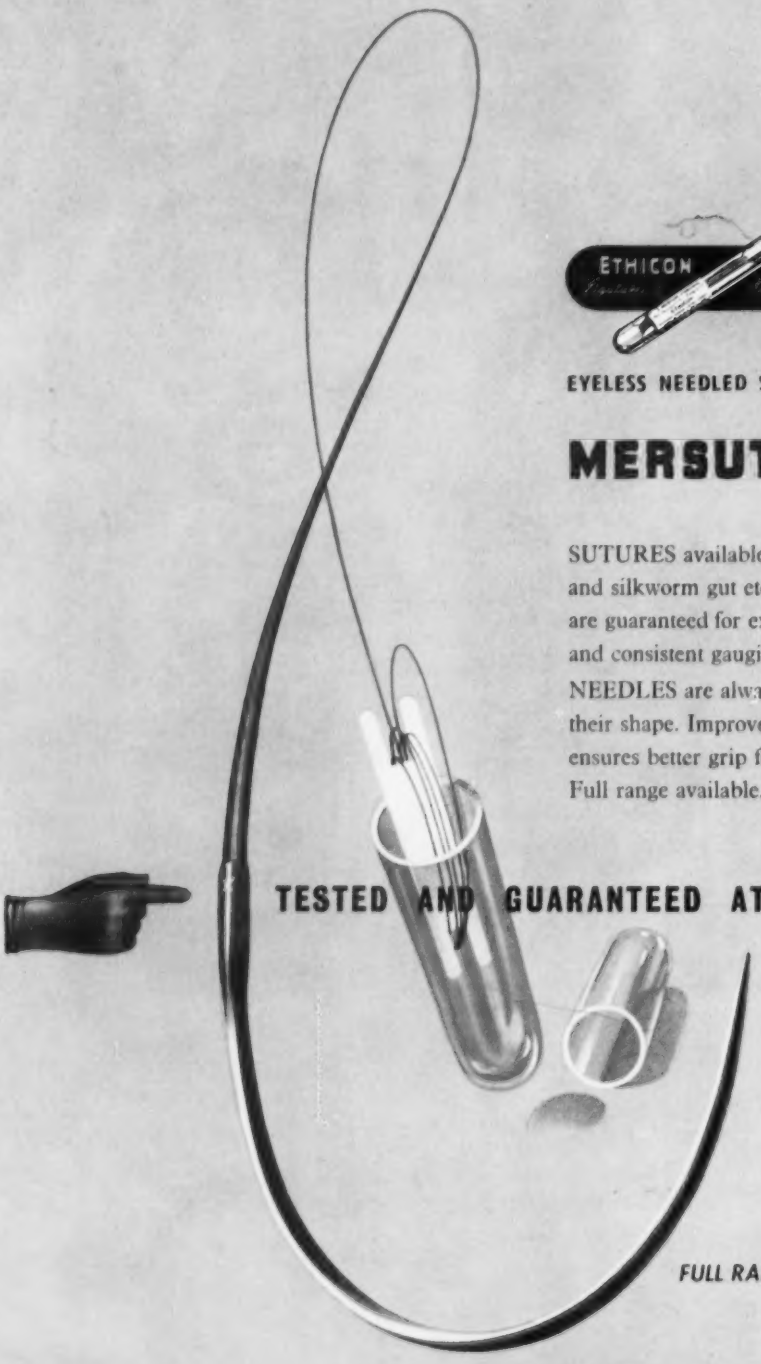

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